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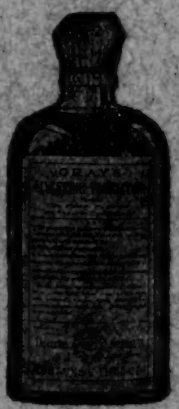

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ANNALS *of* SURGERY

Vol. LXXXIX APRIL, 1929 No. 4

PULMONARY INFARCTION AND EMBOLISM*

By Charles E. Farr, M.D.

AND

Rose Spiegel, M.D.

OF New York, N. Y.

A CLINICAL AND STATISTICAL STUDY FROM THE FIRST SURGICAL DIVISION (CORNELL) OF THE NEW YORK HOSPITAL

The border line between the normal and abnormal grouping of the cellular elements of the blood is ill defined. The mechanical factors of stasis and trauma, however slight in degree, whether accidental or due to surgery, must inevitably lead to thrombosis and probably, in many instances, to embolism and minor degrees of infarction. Only the grosser manifestations give clinical evidences and are at times recognized as such. Chemical alterations of blood serum and tissue juices, of which we know very little, must be extremely frequent and unquestionably are important elements in thrombosis.

In this paper, a large series of case histories, both medical and surgical, and covering a wide range of injuries and diseases, were studied with a view to finding evidences of thrombosis, embolism and infarction. The autopsy records have been compared with the clinical data. Tentative conclusions have been drawn, but the subject is as yet far too obscure for exact deductions.

Method of Study.—The records of the First Division of the New York Hospital have been culled for items of value in this problem. Pulmonary embolism, generally recognized as a surgical complication, has been considered from the medical point of view as well. Its medical incidence has been investigated also in those diseases in which it might occur as an unidentified complication: typhoid fever, chronic cardiac valvular disease, lobar pneumonia, tuberculosis, and leukemia. We are very grateful for the coöperation of the First Medical Division in this matter and especially for the interest of Dr. Lewis A. Conner, its chief.

The study includes cases extending back to 1881. As would be expected, their distribution becomes more sparse as the period under consideration becomes more remote. A detailed statistical study, as presented in the tables, extends from 1917 to 1927, during which interval there has been improvement in the case records. This period of ten years is adequate as a basis for statistical deductions.

The errors in this study depend on the impossibility of evaluating the personal equation entering into observation and technic—clinical and pathological. The diagnosis arrived at by autopsy has been taken as exact, and it is obvious how important is the individual interest of the pathologist in

* Read before the New York Surgical Society, November 28, 1928.

finding the thrombus or embolus. Other criteria used are the röntgenological and the clinical diagnosis, and, finally, the description of symptoms which were otherwise designated, but which are more appropriately considered under pulmonary embolism and closely related phenomena. To this end, the complications, bronchopneumonia, lobar pneumonia, and pleurisy, have been investigated.

Pathology.—Pulmonary embolism, in this study, has been considered the metastasis of suspended abnormal elements in the blood, via the right heart, to the lesser circulation, with the resultant effects due to impaction in a pulmonary artery.

This definition includes in its scope a wide range of diverse clinical and etiological phenomena. Of the various embolic possibilities, we shall limit our discussion to thrombi on the venous side of the circulation, including the right heart.

Thrombi vary greatly in mode of formation, characteristics, and clinical significance. A non-friable, or organized, thrombus is not very dangerous, though it may cause inconvenient local occlusive symptoms. Usually, only the disastrous concomitants of thrombosis are considered; but the conservative value of the process should be remembered. The surgeon who dreads postoperative femoral or iliac thrombosis also dreads the alternative of absence of thrombosis—uncontrollable bleeding. Thrombosis in small blood vessels about a site of inflammation is a barrier against dissemination of the infection. Thrombosis of the prostatic and pampiniform plexuses is almost physiological in old age. These are truisms of pathology.

Since thrombosis is the usual event in infection and following surgical procedures, and is frequent in the elderly, why are not embolic phenomena more common? Essentially, the explanation seems to lie in those factors governing the organization and friability of thrombi.

The following is the classical summary of the etiology of thrombi,[1] without which consideration of embolism and thrombosis is not complete: (1) Changes in the blood plasma and formed elements; (2) Changes in the blood stream—rate of flow and vortices; (3) Changes in the vessel wall.

Changes in the Blood.—Coagulation thrombi depend on the release of fibrin by interaction of fibrinogen, prothrombin, and calcium salts in the plasma with thrombokinase from injured leucocytes, blood platelets, or tissue cells. Also, following repeated hæmorrhage, there is increased coagulability. The detritus of red blood cells resulting from hæmolysis serves as a focus of origin for a thrombus.

Obviously, trauma predisposes to thrombosis by the release of thrombokinase from the injured tissue cells.

Agglutination thrombi form by the increased agglutinative properties of red blood cells, as in typhoid fever. (In typhoid fever, our studies and Dr. L. A. Conner's[2] on thrombophlebitis indicate a closer relationship between thrombosis and phlebitis than between thrombosis and agglutination.)

Changes in the Blood Stream.—Stagnation thrombi form because of local stasis above venous valves, in varices, and aneurisms, and in the cardiac

chambers. These thrombi are apt to be of mixed type and resemble post-mortem clots. Stasis alone is not sufficient to cause thrombosis, as Baumgarten's experiments have shown that careful ligation of vessels is not followed by thrombosis, and Welch [3] found that even coarse ligation is not invariably followed by thrombosis. The other factors are apparently more important.

In the formation of marantic thrombi, besides stagnation, the changed composition of the blood and the underlying toxic factors are causative. Only unusually are the very large veins primary seats of marantic thrombi.

The importance of vortices was emphasized by von Recklinghausen. There is an "eddying movement of the outer lines of flow of the blood stream when there are counter currents, or when the blood with retarded flow passes from smaller into larger channels or over obstructions, or especially into spaces relatively too wide for the received volume of fluid." Such irregularities occur in the dilatations just above the insertion of the venous valves and in the femoral vein near Poupart's ligament, which, because of fixation to fascia, cannot adjust itself to a changed volume of blood.

Changes in Vessel Wall.—According to the accepted theories of thrombosis and coagulation, the normal intimal cells secrete an antithrombin. When injured, their inhibitory product is diminished and thrombokinase is released. Be that as it may, phlebitis almost invariably is accompanied by thrombosis. Which is the prior process is undetermined, for simple degenerative changes in the endothelium, and arteritis, with lesions analogous to phlebitis, seldom are accompanied by thrombosis in the general circulation. In arteritis, the force of the blood current is preventive.

In some cases of typhoidal "phlebitis" where it has not been possible to demonstrate the infective organism locally, a subendothelial collection of lymphoid tissue has been found, which may have initiated the thrombosis.

Thrombosis at the site of phlebitis is conservative by keeping the products of inflammation and degeneration from entering the blood stream.

* * * * * *

The changes directly involving the thrombus influence its probable embolism. Besides autolytic degeneration by the leucocytes in the thrombus, and fatty degeneration, the degree and kind of inflammatory reaction of the intimal cells are important. Minimal proliferative action, as in marantic thrombosis, so that the thrombus is loosely attached, and a predominatingly suppurative process in the vessel wall, so that the thrombus disintegrates, are most likely to release a train of embolic phenomena. Cicatrization by the intimal cells, or canalization by central softening and peripheral organization, is most favorable.

The site of thrombosis also influences embolism, as has been indicated in the discussion of vortices. The hyaline thrombi in the pulmonary capillaries in lobar pneumonia are not likely to be dislodged, nor is a thrombus in a small vein which is not subjected to much force from the blood current.

In a larger vein, a beginning thrombus, consisting only of a framework of platelets and leucocytes, is conceivably readily fragmented, particularly by

the changing pressure due to venous pulsations. A constantly fragmenting thrombus could be responsible for pneumonic symptoms in the early part of the post-operative course and might presage the dislodging of a large portion when thrombosis had proceeded to the stage of addition of the cohesive fibrin. According to Welch, cicatrization of a thrombus requires about a week. A thrombus is particularly menacing, when, by accretion of proximal coagulated stagnant blood, it juts into a larger lumen at the point of junction of a tributary, the force of whose blood stream breaks off the projecting part.

Aschoff has pointed out, that, besides the proximal part of the femoral vein where large valves are present, the pelvic plexus is a favorite site. He emphasizes the importance of continued excessive pressure on the wall of a vein—as in the veins of the legs when the body is upright, and on the pelvic plexus by the downward pressure of intestines. When the body is prone—a position negligible in most major surgery—the femoral veins are compressed against Poupart's ligament. In the supine position—which is more important—there is compression of the left iliac vein by the right iliac, middle sacral, and left hypogastric arteries. However, in abdominal surgery, thrombosis most frequently originates in the left femoral vein.

Thrombosis has been attributed to the action of bacteria. Do the bacteria settle in an already formed thrombus? Do they cause changes in the intima primarily which are then followed by thrombosis? This is the problem in thrombophlebitis and also in marantic thrombi. In the latter, special technic has frequently demonstrated the presence of microörganisms. Recently, Rosenow [4] isolated a diplostreptococcus of low virulence from post-operative thrombi with evidence that it is causative of thrombosis. Often the bacteria found in the thrombus are not those of the original disease. What is the train of events here?

In the surgical cases, the introduction of aseptic technic has apparently not been followed by a proportional diminution in the number of cases of post-operative pulmonary embolism.

Thrombosis may occur at a site distant from the infected operative field, as in an appendectomy, complicated by fatal pulmonary embolism, with the thrombosis limited to the larger veins of the left side. On the basis of a theory of infection, it is difficult to account for localization of infective material from the blood vessels of the appendiceal area—portal circulation—in the general caval circulation.

The emphasis on the etiology of thrombosis has been shifted to mechanical conditions during and after the operation. Stasis due to the position during operation has been cited; however, the femoral vein is not compressed and the downward pressure of the intestines is diminished in the supine position. There is far more strain and circulatory embarrassment in the erect position.

The onus has been laid at the door of surgical technic. In a ten-year period, of 12,813 operations performed under blunt dissection, there were twenty-two fatal cases of pulmonary embolism, or .171 per cent., against a parallel period with 12,615 operations under fine dissection with twenty-one

fatal cases, or .167 per cent.; this discrepancy of about 2 per cent. is within the permissible range of statistical error. Compression of blood vessels, defective ligation, unnecessary traumatization of tissues are, perforce, poor surgical technic from any point of view, and presumably do not occur in this service.

The pre-operative preparation of the patient has also been attacked, with particular emphasis on the elimination of foci of infection. However, this is obviously an unattainable ideal. In emergencies, for instance, it is out of the question.

Increase of body fluids is the most practicable procedure suggested, but obviously does not strike at the root of the matter.

The theory that the post-operative pneumonitides have an embolic basis has been gaining ground. Respiratory affections form the greatest single group of complications, besides wound infections, attributable to the operation. The following table shows the partition of complications:

July, 1914–1927

Total Post-operative Complications	Respiratory Complications						
	Abscess of lung	Lobar pneumonia	Broncho pneumonia	Acute bronchitis	Pleurisy	Pulmonary infarction	Pulmonary embolism
Duodenal ulcer..... 48	–	5	2	–	1	–	1
Gastric ulcer..... 18	–	–	3	–	–	–	1
Acute cholecystitis..... 5	–	–	1	–	–	–	–
Appendicitis.....342	–	16	11	–	2	8	6
Hernia: non-strangulated.170	1	15	10	8	1	8	6
Hernia: strangulated..... 19	–	2	2	–	–	1	1
Varix of veins of leg..... 11	–	–	–	–	–	–	2

Determination of the causative factors underlying the post-operative pneumonias would alter only slightly the treatment of the so-called bronchopneumonias, lobar pneumonias, and pleurisies. The therapeutic problem thus is forced back to thrombosis.

Analysis of Autopsied Cases of Pulmonary Embolism.—Before entering this doubtful province of the embolic nature of early post-operative respiratory complications, we shall present an analysis of sixteen autopsied cases, fifteen of which are undoubtedly pulmonary embolism, with practically complete clinical, X-ray, and pathological data. With the deductions which we are enabled to make, we shall analyze the unautopsied material.

The cases of pulmonary embolism which come to autopsy generally present startling symptoms with immediate fatality. The following case summary is typical:

Three days following a diagnosis of bronchopneumonia, based on physical signs and X-ray findings, the patient suffered "a grinding precordial pain, immediately followed by a convulsion. The pulse became imperceptible; the respiration slow and labored. The patient was cyanotic. Death in five minutes."

Whenever the relationship of circulatory to respiratory cessation has been noted on the charts, the former apparently occurred first.

The following is the incidence of this type of case in autopsy material:

Autopsy Record of Pulmonary Embolism

1917–1926—Total number autopsies	1,116	
Total number pulmonary embolism (fatal)	30	
Per cent. of total number	2.68	per cent.

Year	Number of autopsies	Surgical cases	Medical cases	Accident	Per cent. of autopsies
1917	121	3	1	–	3.3
1918	79	–	–	–	–
1919	77	–	–	1	1.28
1920	87	–	–	–	–
1921	74	2	–	–	2.7
1922	95	4	–	–	4.2
1923	119	7	–	–	5.8
1924	166	3	1	–	2.4
1925	148	3	–	–	2.03
1926	150	5	–	–	3.3

Following is a summary of the autopsied surgical cases available for our examination.

TABLE I.

Analysis of Fifteen Autopsied Cases of Pulmonary Embolism.

CASE 1.—Female, aged fifty-seven years. Suddenness of death—immediately. Operation—perineorrhaphy. Post-operative day onset—seventeenth. Previous symptoms—sixteenth post-operative—dyspnœa. Pathological report: Pulmonary artery—large thrombus; source of embolism—left femoral and three left iliac veins.

CASE 2.—Female, aged forty-two years. Suddenness of death—twenty-five minutes. Operation—exploratory laparotomy, appendectomy, cholecystectomy, bilateral salpingectomy. Post-operative day onset—eleventh. Previous symptoms—remittent 99°–101°. Pathological report—pulmonary artery—all branches. Source of embolism—left internal iliac vein.

CASE 3.—Male, aged thirty-four years. Suddenness of death—three days agony: five minutes. Operation—appendectomy (chronic). Post-operative day onset—fifteenth. Previous symptoms—bronchopneumonia, third clinical and X-ray. Pathological report—pulmonary artery—pulmonary artery and branches. Lungs—infarcts bases. Source of embolism—right internal iliac branch; left internal veins. Miscellaneous—*B. coli* in thrombus.

CASE 4.—Female, aged fifty-nine years. Suddenness of death—ten minutes. Operation—radical amputation of breast. Post-operative day onset—eighth. Previous symptoms—remittent 98°–101°. Precipitating cause—fainted in lavatory. Pathological report—pulmonary artery—pulmonary artery and branches. Source of embolism—disintegrated axillary vein.

CASE 5.—Female, aged fifty-five years. Suddenness of death—two days agony, twenty minutes. Operation—ventral herniotomy. Post-operative day onset—seventeenth. Previous symptoms—bronchopneumonia two days. Pathological report—pulmonary artery—pulmonary artery and branches. Lungs—infarcts bases. Source of embolism—from internal profunda femoris into femoral vein. Miscellaneous—infected laparotomy wound.

CASE 6.—Male, aged fifty-one years. Suddenness of death—eight hours agony, five minutes. Operation—appendectomy (gangrene free pus). Post-operative day onset—fifth. Previous symptoms—undiagnosed. Pathological report—heart—chronic myocarditis. Pulmonary artery—left pulmonary artery. Lungs—right base. Source of embolism—no examination.

CASE 7.—Male, aged thirty-four years. Suddenness of death—thirty minutes. Operation—appendectomy (chronic). Post-operative day onset—ninth. Precipitating cause—sitting up. Pathological report—heart—large thrombus right ventricle. Pulmonary artery—large at bifurcation of pulmonary artery. Source of embolism—left internal iliac vein.

CASE 8.—Male, aged forty years. Suddenness of death—five minutes. Operation—exploratory laparotomy and gall-bladder drainage. Post-operative day onset—first. Previous symptoms—on admission dyspnœa and cyanosis. Pathological report—heart—chronic myocarditis. Pulmonary artery—pulmonary artery and across bifurcation. Lungs—infarct left base. Miscellaneous—acute gastric dilatation.

CASE 9.—Male, aged forty-seven years. Suddenness of death—five minutes. Operation—fractured ribs—no operation. Post-operative day onset—second in hospital. Previous symptoms—bronchitis—103°. Pathological report—heart—dilatation right ventricle. Pulmonary artery—pulmonary artery and across bifurcation. Lungs—congested bases right areas hæmorrhage. Source of embolism—local injury.

CASE 10.—Male, aged sixty-three years. Suddenness of death—three hours. Operation—exploratory thoracotomy for subphrenic abscess. Post-operative day onset—fifteenth. Previous symptoms—fifteenth—apparent heart failure. Pathological report—pulmonary artery—both branches. Miscellaneous—suppurative cholecystectomy.

CASE 11.—Male, aged fifty-four years. Suddenness of death—seven days. Operation—repair of perforated ulcer. Post-operative day onset—thirteenth. Pathological report—heart—verrucous endocarditis. Pulmonary artery—left. Lungs—gangrene left. Source of embolism—thrombophlebitis right internal iliac vein. Miscellaneous—from peritonitis, staphylococcus, and streptococcus hæmolysis.

CASE 12.—Male, aged fifty-five years. Suddenness of death—four days agony, thirty minutes. Operation—bilateral herniotomy. Post-operative day onset—fifth to ninth. Previous symptoms—severe cough, pleural pain on fifth. Pathological report—pulmonary artery—both branches. Lungs—congested. Source of embolism—left external and internal and common iliac veins.

CASE 13.—Male, aged sixty-four years. Suddenness of death—five minutes. Operation—suprapubic cystostomy local anus. Post-operative day onset—tenth. Precipitating cause—enema. Pathological report—heart—large thrombus right ventricle. Pulmonary artery—both branches. Lungs—no change. Source of embolism—right and left internal iliac veins. Miscellaneous—from heart streptococcus non-hæmolysis.

CASE 14.—Male, aged sixty-five years. Suddenness of death—ten days. Operation—first stage—perineal prostatectomy, local anus. Post-operative day onset—twenty-seventh. Previous symptoms—lobar pneumonia X-ray—thirty-second not characteristic middle right lobe. Thirty-seventh dilatation right ventricle and super vena cava including density in right base. Pathological report—heart—dilatation right ventricle. Pulmonary artery—smaller branches. Lungs—right middle lobe congested. Source of embolism—prostatic plexus right internal iliac vein.

CASE 15.—Male, aged thirty-six years. Operation—fractured ribs and acetabulum. Post-operative day onset—third. Pathological report—pulmonary artery—main and left. Source of embolism—left internal iliac vein. Miscellaneous—pelvic tissue necrotic.

Physical examination on admission in no case revealed any cardiac or pulmonary abnormality.

The above Table I indicates the following:

1. The age range is between thirty-four and sixty-five—average fifty years.

2. Average day of onset of fatal symptoms, eleventh, limits first to twenty-seventh.

3. Partition of cases among operations: Appendectomy, 3; herniotomy, 2; exploratory laparotomy, 2; prostatectomy, 2; perineorrhaphy, 1. This distribution is inconclusive.

4. Relation of site of thrombosis to operative field:

(1) Perineorraphy: Left femoral vein; internal iliac; external iliac; left common iliac.

(2) Exploratory laparotomy: Bilateral salpingectomy; cholecystectomy; appendectomy; plication of round ligaments; lipectomy; left internal iliac vein.

(3) Appendectomy: Left internal iliac vein—small branches; right internal iliac vein—up to right common iliac.

(4) Radical amputation—right breast: Right axillary vein—disintegrated.

(5) Repair of ventral hernia: Left profunda femoris vein and projection into femoral vein.

(6) (No examination.)

(7) Appendectomy: Left internal iliac vein.

(8) (No examination.)

(9) Exploratory laparotomy: Origin not found.

(10) Fractured right ribs: Local injuries—partial collapse of lung.

(11) Thoracotomy: Origin not found—no phlebitis.

(12) Repair of perforated duodenal ulcer: Thrombophlebitis right internal iliac vein.

(13) Repair of bilateral hernia: Left internal, external, and common iliac veins.

(14) Prostatectomy, first stage: Both internal iliac veins.

(15) Prostatectomy: Prostatic plexus, right internal iliac vein.

(16) Fractured acetabulum and ischial spines; fractured ribs; dislocation of hip: Left internal iliac vein and branches (necrotic pelvic tissue).

(17) Suprapubic cystostomy: Septicemic infarction.

5. Frequently warning pulmonary signs and symptoms are present, though the final death agony is brief; of the fifteen cases seven lasted more than an hour; in five, it was a matter of days.

6. Four cases were septic—Nos. 2, 4, 5, 11. In all but No. 2, symptoms lasted several days.

7. Clinical diagnosis of pulmonary embolism in its prodromal stage is frequently erroneous, or, perhaps more accurately stated, is misleading in terminology.

8. X-ray findings of a newly-lodged embolus, or a newly-formed infarct, are misleading, principally because of the desire of the clinician to receive a positive, clear-cut statement. Obviously, the anatomical changes following a process of one or two days, or occasionally several hours' duration, cannot be

more than suggestive. In case No. 3, "left bronchopneumonia" was found at autopsy, two days later, to be a typical wedge-shaped infarct in left lower lobe.

9. The type of anæsthesia is apparently of no significance. Case Nos. 13 and 14, two elderly men with enlarged prostates, who were in very good condition on admission, were operated on under regional anæsthesia.

10. The importance of pulmonary embolism as cause of sudden death is variable.

1. Case No. 8, on admission, had the same symptoms which were present at death; severe dyspnœa and cyanosis. The infarct in the left lower lobe was inadequate as a cause of death from cardiac embarrassment and from encroachment on the margin of safety of lung tissue. The chronic myocarditis and the acute gastric dilatation were probably the lethal factors.

2. Case No. 11. Details of the history indicate that on the first day of symptoms patient suffered an attack of pulmonary embolism; "sudden, terrific pain in right chest with immediate collapse, coughed up bloody sputum and had moist râles and impaired resonance over right base. Three days later, friction rub in left axilla, and bronchial breathing in left base. Several days later coughed up black, foul, purulent sputum." Autopsy revealed pulmonary gangrene of left upper lobe, secondary to an infected embolus from the thrombophlebitic right internal iliac vein, as the most probable cause of death.

3. Case Nos. 2, 4, 5, 12. When the pulmonary artery or its first subdivisions are occluded by an embolus, profound shock and rapidly failing circulation ensue, due, perhaps, to the sudden ischemia, or to the irritation of the efferent nerves of the vessel wall by the changed circulatory conditions. In no case do we know how much of the occluding mass is the original embolus, nor about how much time was required for the surrounding thrombus to form. A small embolus may block a minor artery and part of the column of blood toward the heart, thrombosed because of stasis. The original embolus may be responsible for prodromal symptoms culminating, several hours later perhaps, in the terminal agony when a major branch, or the pulmonary artery itself, becomes occluded. The problem is to correlate the clinical and pathological sequence.

4. A large thrombus of embolic origin in the right ventricle may interfere with the flow of blood enough to cause death. Case Nos. 7 and 13. Death occurred before there was any tissue reaction to the emboli lodged in the branches of the pulmonary artery, so that the sudden death may be attributed to right heart failure.

5. The disturbed circulation may embarrass a heart with chronic myocarditis, and dilatation of the right ventricle occur. Case Nos. 8, 10, 14, 15—in at least six of the cases, therefore, cardiac involvement was a factor in producing death. Particular care of the surgical patient with myocardial disease is, therefore, indicated.

11. In regard to diagnosis, there were several illuminating errors.

A female, thirty years of age, whose chief complaint was epigastric pain and whose physical examination was absolutely negative, was operated upon for probable cholelithiasis and chronic appendicitis. No pathological condition adequate to account for the symptoms was found. On the second post-operative day, while talking to a neighbor, the patient "stopped, made a little sound, and became unconscious. Pulse imperceptible. Died in a few minutes." Death was attributed to pulmonary embolism. The only significant finding at post-mortem examination was a stenosed right coronary artery and luetic aorta. The record of this case, besides indicating that the entire clinical picture was due to coronary sclerosis, demonstrates that death due to pulmonary embolism has some clinical features in common with sudden cardiac death. Similarly in the following case: Man of eighty-five years, with chronic bronchitis and myocarditis. At autopsy, a staphylococcus aureus lobar pneumonia and coronary sclerosis were found, which were the probable causes of death.

12. The precipitating cause frequently is unassignable and practically always is undetermined.

Case No. 7.—It does not seem, when matters are in such a precarious state, that sitting up in bed is sufficient to start a thrombus on its embolic career, that immobilization would stave off the evil day. It may very well be that the restlessness was merely symptomatic rather than causative. In looking for a precipitating cause, the tendency is to use the *post hoc, ergo propter hoc* principle.

Case No. 13.—Death occurred during the administration of an enema. The frankly ante-mortem thrombus found in the heart dates the lodging of the embolus at least twenty-four hours before death; therefore, the enema could not have precipitated the embolism.

13. Clinically none of these cases manifested any evidence of venous thrombosis, extensive as was the process in most cases. (This may be explained on the following basis: The large deep veins draining the lower extremities have an adequate collateral circulation, while the smaller and more superficial have not. Occlusion of the latter, therefore, more frequently causes signs.)

14. Case No. 12 suffered two attacks of pulmonary embolism on the fifth and ninth post-operative days.

Analysis of Unautopsied Cases of Pulmonary Embolism.—In this series of twenty cases diagnosed clinically, we consider only those in which there is an episode of sudden collapse, dyspnœa, and pleural pain during convalescence. The milder cases, lacking the symptom-complex of collapse, are considered under the heading "Pulmonary Infarction." In some instances, it is difficult to determine from the case records how severe the onset was. Some of the cases are on the border line, part of them has been included in this group, and others in Pulmonary Infarction; so that the error has been equally distributed.

In the decade 1917–1926, there were thirteen autopsied cases, nine unautopsied deaths, and seven recoveries, the chances for recovery thus being twenty-five per cent., to place the emphasis optimistically. The following case summary (Table II) includes all the records we have been able to obtain beyond the above decade, as well.

Table II.

Analysis of Twenty Unautopsied Cases of Pulmonary Embolism.

Case 1.—Female, aged twenty-eight years. Operation—supravaginal hysterectomy. Post-operative day onset—seventh. Previous symptoms—convalescence good; slight distention. Symptoms of pulmonary embolism—in normal sleep: groaned, restless. Pulse: weak, gasping, pale, cyanotic. Result—died.

Case 2.—Female, aged thirty-five years. Operation—appendectomy and freeing of adhesions. Post-operative day onset—eighth. Symptoms of pulmonary embolism—sudden pain right chest, marked dyspnœa, bloody sputum. Result—lived.

Case 3.—Male, aged forty-six years. Operation—appendectomy (chronic). Post-operative day onset—eleventh. Previous symptoms—pain in right back—râles. Thirteenth day post-operative—symptoms of pulmonary embolism—expired suddenly. Result—died.

CASE 4.—Male, aged twenty-nine years. Operation—herniotomy. Post-operative day onset—fourth. Previous symptoms—sudden pain in left chest and slight consolidation. No pleural rub; X-ray hazy shadow in left. Fifteenth day post-operative. Symptoms of pulmonary embolism—similarly in right chest; X-ray—consolidation left base and right hilum. Result—lived.

CASE 5.—Female, aged thirty-two years. Operation—appendectomy (gangrenous). Post-operative day onset—tenth. Previous symptoms—sudden pain right base; bloody sputum—pleural rub. Precipitating cause—getting out of bed. Post-operative day onset—fourteenth. Symptoms of pulmonary embolism—similarly in right chest. Result—lived.

CASE 6.—Male, aged thirty-two years. Operation—appendectomy. Post-operative day of onset—thirteenth. Symptoms of pulmonary embolism—collapse; pain in chest. Labored respiration. Result—died.

CASE 7.—Male, aged twenty-two years. Operation—division of Harris band. Post-operative day of onset—fourth. Previous symptoms—mushy first sound and apical systolic murmur. Symptoms of pulmonary embolism—pain left chest. Some collapse. Result—lived.

CASE 8.—Female, aged forty-nine years. Operation—excision of varix (in saphenous ring). Post-operative day of onset—eleventh. Symptoms of pulmonary embolism—sudden collapse. Result—died.

CASE 9.—Female, aged twenty-nine years. Operation—appendectomy. Previous symptoms—phlebitis of left saphenous vein; cellulitis. Symptoms of pulmonary embolism—pain left chest; bloody sputum; slight collapse. Result—lived.

CASE 10.—Female, aged forty-two years. Operation—appendectomy. Post-operative day of onset—eighth. Symptoms of pulmonary embolism—pain in chest. Result—lived.

CASE 11.—Female, aged fifty-seven years. Operation—cholecystectomy. Post-operative day of onset—ninth. Symptoms of pulmonary embolism—pain left chest; cough; slight collapse. Result—lived.

CASE 12.—Female, aged forty-three years. Operation—hysterectomy. Post-operative day of onset—eighth. Previous symptoms—varicose veins legs on admission. Symptoms of pulmonary embolism—pain, cyanosis, collapse. Result—died.

CASE 13.—Female, aged thirty-eight years. Operation—hysterectomy. Post-operative day of onset—sixth. Previous symptoms—98°-100° fluctuations. Symptoms of pulmonary embolism—sudden dyspnœa and thoracic pain; nausea; loss of sight and hearing. Result—died.

CASE 14.—Female, aged twenty-five years. Operation—salpingectomy. Post-operative day of onset—twelfth. Previous symptoms—X-ray positive on twenty-fourth post-operative day. Symptoms of pulmonary embolism—pain in right chest; 104°. Result—lived.

CASE 15.—Male, aged forty-one years. Operation—laparotomy (liver abscess). Post-operative day of onset—ninth. Symptoms of pulmonary embolism—dyspnœa; collapse. Result—died.

CASE 16.—Female, aged fifty years. Operation—repair of umbilical hernia. Post-operative day of onset—fifth. Previous symptoms—Pain right chest; slight signs of consolidation in right axilla; bloody sputum. Eleventh day post-operative—symptoms of pulmonary embolism—sudden collapse. Result—died.

CASE 17.—Female, aged fifty-five years. Operation—drainage of pelvic abscess. Regional anæsthetic. Post-operative day of onset—second. Previous symptoms—post-operative vomiting. Symptoms of pulmonary embolism—convulsions; pulse 100. Respiration; fall in blood pressure; coma. Result—Died.

CASE 18.—Female, aged forty-seven years. Operation—drainage of gall-bladder. Post-operative day of onset—tenth. Previous symptoms—Severe saphenous phlebitis (varicose veins on admission), convulsive attack. Symptoms of pulmonary embolism—(assoc. chronic myocarditis). Fourteenth day post-operative—Symptoms of pulmonary embolism—dyspnœa, cyanosis, vertigo, apprehension, collapse. Result—died.

CASE 19.—Male, aged thirty-eight years. Operation—gastro-enterostomy. Post-operative day of onset—first. Previous symptoms—pain in chest, 103°. Second day post-operative—symptoms of pulmonary embolism—106°; cough; pain; collapse. Result—died.

CASE 20.—Female, aged forty-two years. Operation—herniotomy. Post-operative day of onset—eighth. Previous symptoms—post-operative acetonuria. Symptoms of pulmonary embolism—sudden pain right chest; cyanosis; faint. Result—lived.

1. Age range—twenty-two to fifty-five years; average—thirty-nine years.
   Average age in fatal cases .....forty-two years.
   Average age in recoveries .....thirty-five years.

In comparing these figures with similar ones from the autopsied cases, one is struck by the definitely younger age group above and with the younger group in the recoveries.

2. Average day of onset—seventh post-operative (frank symptoms of pulmonary embolism). Six out of twenty occurred in the first week. Note the earlier time of onset in this series.

3. Five cases (Nos. 3, 4, 5, 16, 19) apparently suffered more than one attack—three deaths; recoveries were in the youngest individuals. In Case Nos. 4 and 16, physical examination at the time of onset of pleural pain revealed slight signs of consolidation and no pleural rub. The pathological process must have antedated the symptom of pain. We are accustomed to thinking that the pain is practically simultaneous with the embolic impaction.

4. CASE No. 18.—This case with definite cardiac involvement had an ample opportunity for the development of pulmonary embolism.

5. One recovered case and one fatal case had saphenous phlebitis, Case Nos. 9 and 18.

6. Of conditions predisposing to pulmonary embolism:
   Two—cardiac involvement—Case Nos. 7 and 18 which were fatal.
   Two—varicose veins—Case Nos. 8 and 12 both were fatal.

7. Partition among operations: Appendectomy, 6; gynæcological operations, 5; cholecystectomy, 2; hernial repairs, 2.

8. The following case summary we believe to be another instance illustrating some of the diagnostic pitfalls:

Male, fifty-five years of age, with a strangulated left indirect inguinal hernia, of negative physical examination, but for the local condition was operated on under local anæsthesia. The mesentery of the colon showed some thrombosed vessels, but seemed viable. On the morning of the third post-operative day there was sudden collapse, the temperature rose from 101° to 103°. The patient suffered a chill and became cyanotic. He had severe cramp-like abdominal pains. He recovered from the collapse to suffer a fatal one at 6 P.M. During the post-operative course, the pulse gradually increased from 70 to 140 and the respiration from 28 to 48, but no physical signs to account for this had been noted, apparently. Death was attributed to pulmonary embolism. The circumstances indicate death due to mesenteric thrombosis as more probable. Here, the common factor of collapse was the source of confusion.

Pulmonary Infarction.—Pulmonary infarction, roughly speaking, is characterized by some of the following symptoms and physical signs: Pleural pain, mild distress, discomfort in breathing, cough, frothy sputum with an admixture of bright or clotted blood, sharp rise in temperature, friction rub, small area of slight consolidation, slightly diminished resonance, harsh or bronchial breathing, fine râles.

This syndrome requires a different emphasis on its etiological factors from the two types already discussed. Here we must consider abnormal local conditions in the lung, the effect of the ether anæsthesia, the infectivity of the thrombus, as well as the mechanical effect of the impaction of the embolus.

The collateral circulation of pulmonary, pleural, and bronchial vessels normally is adequate to compensate for any obstruction in a minor pulmonary artery. Arterial anæmia in the area of distribution causes collateral hyperæmia of adjacent arteries followed by immediate hyperæmia of the site and then by equalization. However, if the part already is in a condition of venous engorgement and high pressure, the collateral hyperæmia increases the pressure, the capillaries dilate to maximum, and diapedesis of red cells occurs. Thus, obviously, any factors contributing to cardiac embarrassment, for instance emphysema, are important at operation. The degree to which ether increases pulmonary secretion must be considered a factor in increased intrapulmonary pressure, predisposing to infarction.

Given this underlying condition, the manifestation may be pyæmic—a septic pneumonia, abscess, or gangrene. Bland infarcts are accompanied very frequently by some pleural effusion, which may be due to maladjustment of the blood supply, or to a reactive pneumonia and surrounding localized œdema. Rarely, the infarct may be anæmic, if the circulation is very poor.

The above pathological summary is the rationale for considering that the post-operative pneumonitides and pleurisies have an embolic basis, coupled with the fact that thrombosis is frequent post-operatively.

We append fifteen case summaries of post-operative pulmonary infarction as typical of the uniformity and variation in the findings. This does not include our entire array of cases which we shall summarize very briefly elsewhere.

1. In this table (III)—average age—thirty-two years. Age range—fifteen to forty-nine years.

2. Average day of onset of frank signs post-operatively—seventh. Nine out of fifteen in the first week.

3. One death—Case No. 12.

4. Phlebitis—Case Nos. 6, 8, 9. No fatalities.

TABLE III.

Analysis of Fifteen Cases of Post-operative Pulmonary Infarction.

CASE 1.—Male, aged forty-nine years. Operation—appendectomy gangrenous—local peritonitis. Post-operative day of onset—eleventh. Symptoms of infarction—severe pain in right lower chest; slight fever.

CASE 2.—Male, aged forty-five years. Operation—herniotomy. Post-operative day of onset—tenth. X-ray—opacity left middle chest. Symptoms of infarction—rheumatic pain in left shoulder; following day—bloody sputum.

CASE 3.—Male, aged sixteen years. Operation—appendectomy. Post-operative day of onset—fifth. X-ray—congestion of right upper lobe. Symptoms of infarction—sudden—106°; no symptoms; slight consolidation upper right chest.

CASE 4.—Female, aged forty-five years. Operation—excision of varicose veins of right leg. Post-operative day of onset—tenth. Symptoms of infarction—pain in right chest; sore throat; cough and pain in left chest.

CASE 5.—Male, aged fifteen years. Operation—tonsillectomy. Post-operative day of onset—twenty-first. Symptoms of infarction—awakened by sharp pain in chest; increased by respiration; dyspnœa. Examination negative. Eleven days after admission fluid posterior left base. Gram-positive cocci.

CASE 6.—Male, aged thirty years. Operation—bilateral herniotomy. Post-operative day of onset—seventh. Symptoms of infarction—pain in chest, cough, fever; slight hemoptysis; consolidation right lung; coarse râles; ten days post-operative a thrombosis in left leg.

CASE 7.—Female, aged twenty-eight years. Operation—myomectomy appendectomy. Post-operative day of onset—twelfth. Symptoms of infarction—pain in right chest; dyspnœa, apprehension; posterior right base consolidation.

CASE 8.—Female, aged forty years. Operation—Billroth No. 1. Post-operative day of onset—twelfth. Previous symptoms—sixth, seventh, ninth, eleventh days unexplained rises in temperature. Symptoms of infarction—seventh day pain in right lower chest and in right knee. Twelfth day bloody sputum.

CASE 9.—Male, aged forty-three years. Operation—appendectomy (peritoneal abscess). Post-operative day of onset—sixth. Symptoms of infarction—pleural rub and pain in left chest. Bronchopneumonia twelfth day, phlebitis right calf. Post-operative day of onset—fourteenth. Symptoms of infarction—pleural rub and pain in right chest.

CASE 10.—Male, aged twenty-eight years. Operation—herniotomy bilateral (acutely inflamed asymptomatic appendix). Post-operative day of onset—second. Symptoms of infarction—104°—consolidation right posterior base; coarse râles; residual râles; sputum positive tuberculosis.

CASE 11.—Male, aged twenty-eight years. Operation—herniotomy right. Post-operative day of onset—third. Previous symptoms—(on admission rhonchi.) Symptoms of infarction—pain in chest; cough; two days later blood tinged, watery sputum; some râles everywhere; increased temperature.

CASE 12.—Male, aged twenty-three years. Operation—herniotomy right. Post-operative day of onset—first. X-ray—resembles new growth. Symptoms of infarction—septic temperature until death sixty-ninth post-operative day; pain and consolidation right chest; puncture; sterile necrotic crumbly material; boils. Signs persisted until death.

CASE 13.—Male, aged twenty years. Operation—herniotomy bilateral. Post-operative day of onset—second. Symptoms of infarction—104°. Left base posterior—consolidation; fine râles; bloody sputum; cough. Next day—dulness; vesicular breathing; temperature normal.

CASE 14.—Male, aged thirty-nine years. Operation—appendectomy. Post-operative day of onset—third. X-ray—fluid right chest. Symptoms of infarction—104°. Pain in right base posterior; impaired resonance and breath sounds; fourteenth post-operative day fluid signs right base.

CASE 15.—Male, aged twenty-eight years. Operation—appendectomy. Post-operative day of onset—first. X-ray—pneumonia left base. Symptoms of infarction—cough, flushed face; dyspnœa—103°; left lower lobe—consolidation.

The noteworthy points are the younger age group, the earlier onset of pulmonary symptoms, the higher incidence of phlebitis, and the lower mortality, as compared to the last series discussed. Perhaps this may be explained as follows: Thrombosis, excited in an unknown manner, does not proceed far, because of the better general circulation in these younger individuals. Bits of the thrombus are washed away and are responsible for the pulmonary symptoms. The force of the circulation prevents the formation of large, more disastrous thrombi. This is purely speculative.

Our series of cases of pulmonary infarction includes ten which were salvaged from other classifications. They present a sufficient number of the characteristic symptoms and signs of pulmonary infarction to come under this heading. It is safe to assume that, had observations been recorded more fully in other cases, more would have come in this category.

Influence of Ether.—As has already been stated, ether anæsthesia may predispose to pulmonary infarction. We have tried to evaluate this factor.

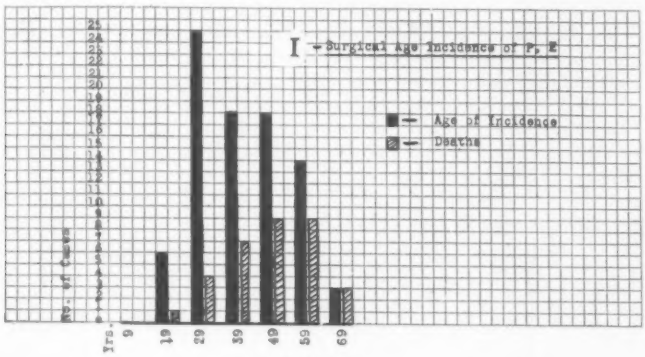
In seventeen of sixty-nine cases, the duration of ether anæsthesia was one hour or over. Obviously, more extensive surgery was required in these cases, and this factor may have been the more predominant cause of infarction. In only five, was there moderate, or much, mucus during the anæsthesia; in no instance corresponding with any of the above seventeen.

During ether anæsthesia the blood pressure, as well as the pulse rate, is normal or increases, so that there is no general circulatory stasis. However, the pressure in the pulmonary artery is raised, and this, as well as any increased intrapulmonary pressure due to bronchorrhea, predisposes to hæmorrhagic infarction. The experimental production of pulmonary infarcts under ether in otherwise normal lungs has been negative.

No truly comparable series of major operations performed under local and under general anæsthesia is possible, because of the grave condition of the patient necessitating the regional anæsthesia. Practically, our study is unilluminating.

General Survey of Surgical Pulmonary Embolism and Infarction.—Total number of cases in the records of First Surgical Division are eighty-four, of which thirty-three were fatal.

The distribution according to age, of the incidence of cases and of the incidence of deaths, is shown in Graph I. This demonstrates strikingly



GRAPH I.

the earlier age involvement and the concomitant lower death rate per early decade of life, than other investigations have led us to believe.

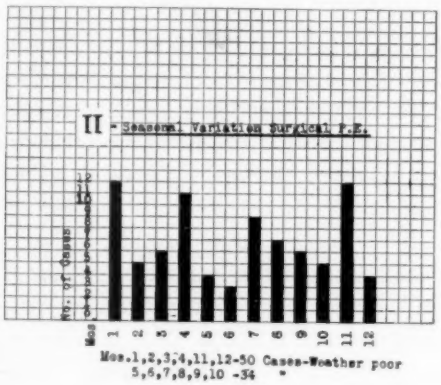
In 1909, Gibson [5] noted the comparative freedom from embolism under forty years of age and the milder train of symptoms in such cases. He also emphasized the fact that most cases occur during the first seven days—60 per cent.—and from intervention "below the belt."

The distribution according to sex—this condition is approximately twice as frequent in males, but the death rate in males is 37.3 per cent., and in females it is 42.4 per cent.

Seasonal variation—cases of pulmonary embolism predominate in the months of unstable weather. (Graph II.)

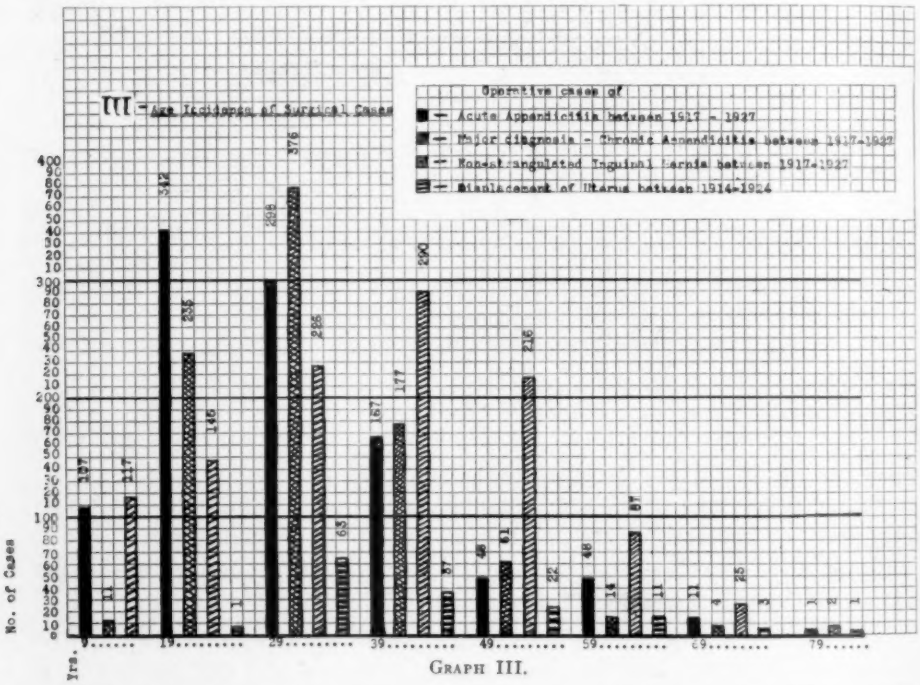
Distribution among operations—1914 to 1927. Appendectomy: 2,962 operations, seventeen cases. Herniotomy: 1,848 operations, twelve cases. (Graph III.)

Examination was made of twenty-nine consecutive cases of prostatectomies between January and May, 1926, with a view to determining unidentified



GRAPH II.

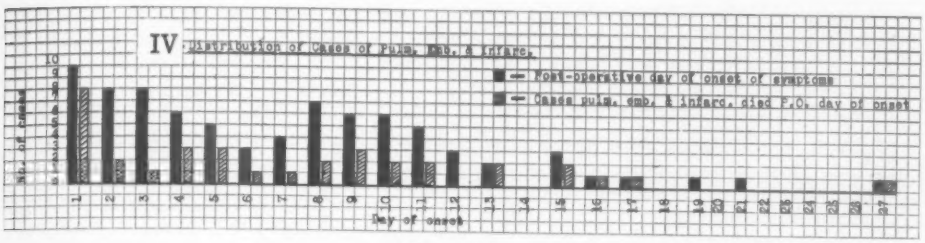
cases of pulmonary infarction and phlebitis: Three cases of phlebitis; one—(nurse's note)—none followed by pulmonary symptoms; six cases of respiratory complications: pulmonary infarction, four; pneumonia (and myocardial degeneration), one; pleurisy, one.


III - Age Incidence of Surgical Cases
Operative cases of
Acute Appendicitis between 1917 - 1927
Major diagnosis - Chronic Appendicitis between 1917-1927
Non-strangulated Inguinal Hernia between 1917-1927
Displacement of Uterus between 1914-1924
No. of Cases
Yrs.

GRAPH III.

Similarly with displacement of uterus and repairs between May, 1918, and March, 1924, there were sixty-nine consecutive cases, of which two had varicose veins without pulmonary consequences.

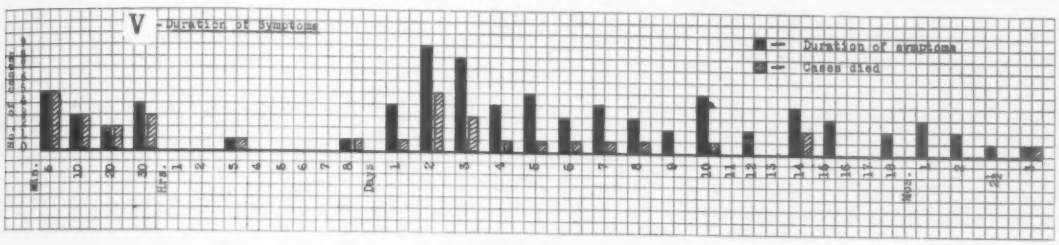
Onset.—Graph IV.—In this table, the onset is considered to date from the first appearance of symptoms, not necessarily of the frank pulmonary embolism and infarction. We wish to emphasize the more subtle overtures of pulmonary embolism. The preponderance of cases in which the onset is in the


IV
Distribution of Cases of Pulm. Emb. & Infarc.
Post-operative day of onset of symptoms
Cases pulm. emb. & infarc. died P.O. day of onset
No. of cases
Day of onset

GRAPH IV.

early post-operative course is marked. Attention has already been called to the progressively younger individuals involved with the earlier onset and more benign course.

Duration of Symptoms.—Graph V.—As a corollary to the recognition of the more benign pulmonary infarction is the recognition of the longer duration and the lower mortality rate accompanying these cases.



GRAPH V.

Temperature, Pulse, and Respiration Changes in Pulmonary Embolism and Infarction.—A temperature over 100° during convalescence has arbitrarily been considered normal.

Change in temperature is the most constant finding, though neither invariable nor uniform; pulse and respiration rates are very much increased in the few cases with immediate collapse, but, in those which recover, soon return to the previous level.

In very few of the cases with a more protracted and less disastrous course of pulmonary symptoms is a change in pulse and respiration rates recorded, though a transitory change may have passed unnoted at the time of onset.

The changes in temperature have been considered under three heads:

1. Those cases having an intermittent, septic temperature during convalescence and before the onset of the pulmonary complications (twenty-four cases);

2. Those in which the fever and onset were simultaneous (twenty-five cases);

3. Those in which there was no change or a delayed change (seventeen cases);

(Unaccounted—fifteen cases).

Group 1.—An irregular, septic, intermittent temperature, 98° to 101°, or 103°. Of the fatal cases, five had definite septic conditions, local peritonitis, appendiceal abscess, subphrenic abscess, etc.

In one case there were unexplained rises in temperature for four days before the appearance of physical signs, probably due to the impaction of four successive emboli.

In the cases in which there was time for the nurses' routine, there was noted a rise in temperature simultaneous with the onset of symptoms, except in one case in which there was a gradual rise to 104°.

The physical signs, when present, and the symptoms persisted longer than the new level of temperature. Resolution by crisis, four; by lysis, six; (others unnoted).

Group 2.—Two cases, which were immediately fatal, suffered a subnormal temperature and other evidences of collapse. Resolution by crisis, seven; by lysis, twelve; persistently high temperature until death, four.

Group 3.—Thirteen cases: No change in temperature, pulse, and respiration, which were within normal range; three cases fatal; four cases septic; three cases sharp rise in temperature—one to three days after frank symptoms or signs; two by crisis, one by lysis.

SUMMARY

The onset of symptoms is generally accompanied by a sharp rise in temperature, lasting less than the symptoms, and unaccompanied by a proportional change in the pulse and respiration level.

Resolution is more frequently by lysis than by crisis. Occasionally there is no change until one to three days after onset of symptoms or none at all.

The convalescent pre-embolic septic temperature may be due to the usual wound complications, or more significantly, to a latent thrombophlebitis.

The prognosis is better in those without a convalescent septic temperature.

The following is the incidence of specific symptoms:

Incidence of Specific Symptoms

Bloody sputum: Autopsied cases, two; pulmonary embolus, four; pulmonary infarction, seventeen; twenty-three out of a total of eighty-four cases.

Signs of fluid: Five dry tap, of which four had no sputum; five positive tap, of which four had no sputum.

Physical signs: Autopsied cases, three; *pleural pain,* 2; asymptomatic, 11. Other cases, 35; *pleural pain,* 45; (of which twenty-five showed both symptoms and physical signs).

X-RAY FINDINGS

Autopsy Findings and Physical Signs.—At the time of the lodging of the embolus, there are no characteristic X-ray features. In only five of the twenty-three X-ray reports was a diagnosis suggestive of pulmonary infarction; in one the X-ray report was positive twelve days after the onset; in another, eight days; in a third, two weeks.

The X-ray report in the first few days of infarction is vague: "Congestion, haziness, or diffuse mottling, and accentuated hilum shadows which suggest a pneumonitis."

In about five days the signs are those of bronchopneumonia, or suggest a pleural effusion. The sequence of changes in the röntgenogram of pulmonary infarction is well shown in the following: "Pneumonitis and congestion in right lung, not a pneumonia." Ten days later, "Massive pleural effusion in lower two-thirds of right chest." Five days later, "Signs of pulmonary infarction or of pneumonia with thickened pleuræ in right chest." Fifteen days later, "Right pulmonary emboli. More likely bronchopneumonia." Two weeks later, "Encapsulated fluid in lower two-thirds of right chest."

This is paralleled by the pathological sequence of compensatory hyperæmia of the collateral circulation, followed by local consolidation and occasionally by pleural involvement, which may be productive or exudative, with, finally, the cicatrization of hæmorrhagic area. Obviously, the wedge-shaped or cuboidal area of pulmonary consolidation, which we expect off-hand in a röntgenogram of diagnostic import, is late in appearing. As in many other instances, the X-ray findings should be viewed in the light of the clinical manifestations. We are taking the liberty of summarizing an excellent discussion on this subject by Wharton and Pierson.[6]

Within twenty-four hours following the onset of the symptoms of pulmonary infarction, the film shows clouding of the costophrenic angle, which also occurs in bronchopneumonia and in pleural effusion. In the former, however, the clinical reaction is disproportionately great in relation to the pathology as demonstrated in the röntgenogram.

In more severe cases of pulmonary infarction, the lung density (in the nature of the lesion, most frequently in the dependent portions of the lung) is increased and is fairly well delimited. Frank pleural changes occur between four and ten days after onset and involve the same area as the pulmonary infarction. They consist of a network of fine lines superimposed on the area of increased pulmonary density and indicative of the formation of bands of thickened pleuræ. The pleural changes persist longer than the pulmonary changes, being a matter of months, very frequently, whereas the latter is a matter of days.

The pulmonary density in infarction differs from that in lobar pneumonia in being less marked and in the absence of a lobar distribution, as a rule.

The X-ray film of pulmonary infarction, as contrasted to that of bronchopneumonia, manifests a more marked pleural reaction and sharper outline of the involved area. In moderate pleural effusion, the shadow is denser and a meniscus is present, while the pleural reaction is slighter. In slight pleural effusion, the diagnosis is more difficult. Clinically, however, the constitutional reaction is not as severe in pleural effusion.

X-ray films of post-mortem cases of pulmonary infarction and embolism might yield profitable information correlating pathological and X-ray manifestations.

PHLEBITIS AND PULMONARY SYMPTOMS

Operation.—Excision of varix of left saphenous vein for phlebitis of left saphenous—(a complication of correction of retroverted uterus).

Venous Symptoms.—Saphenous phlebitis preceding pulmonary symptom tenth post-

operative day. Thrombosis in left leg tenth post-operative day. Pulmonary symptoms—seventh post-operative day. Hæmatoma about spleen following pulmonary symptoms. Phlebitis right saphenous twelfth post-operative day. Pulmonary symptoms—sixth post-operative day. Phlebitis of left calf nineteenth to twenty-sixth post-operative day. Pulmonary symptoms seventh to twenty-first post-operative day. Pain in right knee and right thorax seventh post-operative day. Pulmonary symptoms—bloody sputum twelfth post-operative day. Phlebitis of left saphenous eleventh post-operative day. Pulmonary symptoms—bloody sputum eighth to tenth post-operative day. Phlebitis of left leg eleventh post-operative day. Pulmonary symptoms—bloody sputum nineteenth post-operative day. Pain and swelling in both legs thirteenth post-operative day. Pulmonary symptoms—bloody sputum fourth post-operative day. Phlebitis of both legs. Pulmonary symptoms—infarction right chest. (Time of onset not noted.)

In five cases phlebitis occurred before pulmonary symptoms—two died.

In six cases phlebitis occurred after pulmonary symptoms—one died.

The latter may be explained by the friability of the mural thrombus in the early stage of its formation. While the lumen is still adequate for the blood stream distal to the partial occlusion, we may expect distant embolic phenomena earlier than the local signs and symptoms due to occlusion. That there are more embolic phenomena earlier in thrombosis is apparent from Graph III.

In sixteen autopsied surgical cases of pulmonary embolism, five occurred in the first week post-operative; nine after the first week. In the former group, one suffered from fractured ribs and was not operated on, and another arrived *in extremis* from uncertain cause necessitating an exploratory laparotomy. Clearly in these cases, the causes for embolism antedate hospitalization.

The preponderance of fatal cases after the first week seems to be due to the larger embolus which is dislodged from the more cohesive thrombus present at that time.

Among six cases of phlebitis complicating appendectomies, pulmonary infarction occurred in two—one apparently passing unnoted but for the nurse's record of bloody sputum. In the other cases, pulmonary emboli may have been asymptomatic, granted a normal lung, or the symptoms were unrecorded, or organization of the thrombus occurred *in situ*.

The prognosis appears fair in cases of pulmonary symptoms associated with phlebitis. The reason which some have assigned for this is the attitude of watchful waiting due to the knowledge of the existence of the latter condition. Though this may be a factor where phlebitis precedes, a more important one seems to be the anatomy of the region which becomes obviously involved. In the autopsied cases, thrombosis was on a large scale, spreading from the femoral vein to the iliacs. Thrombosis of the saphenous vein involves a vessel of far smaller lumen and less rapid blood flow so that significant embolic fragments are not dislodged.

DIFFERENTIAL DIAGNOSIS BETWEEN BRONCHOPNEUMONIA AND PULMONARY INFARCTION

Onset and History.—Bronchopneumonia is frequently preceded by a pre-operative bronchitis, etc., or there may be a history of aspiration during

anæsthesia. Pulmonary infarction seldom has a clear-cut history of respiratory involvement, but may be associated with a phlebitis. The onset in bronchopneumonia is insidious. In pulmonary infarction, there may be prodromal symptoms suggesting bronchopneumonia, and a preceding irregular temperature, but there generally—not invariably—is an episode of a sharp rise in temperature. The onset of pulmonary infarction tends to be later than that of bronchopneumonia in the post-operative course.

Pain.—In bronchopneumonia, this symptom is not marked. In pulmonary infarction, this was the most frequent symptom we found. It is marked in its severity.

Sputum.—In both, sputum may be absent. In bronchopneumonia, it is muco-purulent with, perhaps, an admixture of blood. In pulmonary infarction, it is bloody and frothy, the blood being bright red, or clotted.

Respiration.—There is increased rate of respiration in bronchopneumonia. In pulmonary infarction, there may be a transient increase at the onset.

Physical Signs.—These may be the same in both conditions. In bronchopneumonia, it is far more likely that the signs will be diffuse. There rarely is tubular breathing. The breath sounds are harsh; there are crepitant râles.

In pulmonary infarction, the signs are variable—there may be localized friction rubs or crepitant râles lasting two or three days, or consolidation of the lower lobe—localized and not extensive; the signs may be of extensive plastic pleurisy or serous effusion; on exploratory puncture, however, fluid may not be obtained.[7]

Duration of Symptoms and Physical Signs.—Generally, the course in bronchopneumonia is more protracted, and the duration of fever is fairly parallel to the duration of signs. In pulmonary infarction, the temperature subsides considerably before the physical signs or symptoms.

Condition of Patient.—In pulmonary infarction, generally after the first reaction to the onset has subsided, the patient is in fairly good general condition in spite of the pain and expectoration.

MEDICAL CASES OF PULMONARY EMBOLISM AND INFARCTION

Pulmonary embolism and infarction from the medical side do not present a homogeneous class, and include some unique cases. Between 1881 and 1927, thirty-two cases of pulmonary embolism and infarction came to autopsy on the First Medical Service.

The following is the distribution of cases in the entire series: Chronic myocarditis, thirteen; chronic cardiac vascular disease, eight—twenty-two cardiovascular; aortic aneurism, one; lobar pneumonia, three; typhoid fever, two; idiopathic pulmonary infarction, two; tuberculous pleurisy, one; post-partum pulmonary infarction, one; chronic nephritis, one.

CARDIOVASCULAR DISEASE

I. *Autopsied Cardiovascular Cases.*—Examination of post-mortem records in cardiovascular disease complicated by pulmonary infarction reveals several characteristics wherein this type differs from the case on the surgical side.

1. In spite of stasis, which in some cases is generalized, primary thrombosis in the peripheral veins is rare as a source for pulmonary infarction. In this series (Table IV) there is just one case, No. 7, in which this possibility was entertained.

TABLE IV.

Summary of Twenty-two Autopsied Cases of Cardiovascular Disease.

CASE 1.—Male, fifty-three years of age. Disease—cardiac hypertrophy and dilatation. Previous symptoms—*embolic infarcts* (source of embolus indicated): Fragmented thrombi in both ventricles near papillary muscles; emboli in both divisions of pulmonary artery; infarcts—bases.

CASE 2.—Male, sixty years of age. Disease—cardiac hypertrophy and dilatation. Previous symptoms—*embolic infarcts* (source of embolus indicated): Small thrombi in both ventricles; passive congestion of lungs; infarcts—bases; emboli—corresponding arteries.

CASE 3.—Male, sixty-two years of age. Disease—cardiac hypertrophy and dilatation; myocarditis. Previous symptoms—*embolic infarcts* (source of embolus indicated): Coronaries sclerosed—almost occluded; thrombus and fibrous myocarditis—right heart; infarcts and passive congestion of both lungs; pericardial and pleural effusion.

CASE 4.—Male, fifty-nine years of age. Disease—cardiac hypertrophy and dilatation; myocarditis. Previous symptoms—*embolic infarcts* (source of embolus indicated): Thrombi—right auricle; infarcts—all lobes; passive congestion of lungs.

CASE 5.—Female, fifty years of age. Disease—aortic aneurism. Previous symptoms—*embolic infarcts* (source of embolus indicated): Luetic aneurism of arch with lamellated clot; small mural thrombi in right auricle; in right and left pulmonary artery—thrombi extending from right ventricle; left base consolidated.

CASE 6.—Male, forty-five years of age. Disease—arteriosclerotic endocarditis. Previous symptoms—*embolic infarcts* (source of embolus indicated): Torn thrombi in right ventricle; left pulmonary artery—embolus; left lung anæmic; right lung multiple infarcts.

CASE 7.—Male, fifty years of age. Disease—bronchopneumonia; myocarditis (chronic). Previous symptoms—*embolic infarcts* (source of embolus indicated): Syphilitic endocarditis; mural thrombus in left ventricle—about one-half of cavity filled; interference with function sufficient to cause decompensation; infarct left lung—probably from pelvis or leg; bronchopneumonia right base; kidney infarcts.

CASE 8.—Male, fifty-six years of age. Disease—coronary thrombosis; myocarditis (chronic). Previous symptoms (source of embolus indicated): Mural thrombus—right auricle; infarct—recent—left lung—lower; hydropericardium, thorax, peritoneum.

CASE 9.—Male, thirty years of age. Disease—chronic endocarditis; myocarditis (chronic). Previous symptoms—*embolic infarcts* (source of embolus indicated): Mitral and tricuspid vegetations—streptococcus viridans; one branch of right pulmonary artery is occluded by an old thrombus extending into the smaller branches; infarcts—right upper; left lower; pericarditis and pleurisy.

CASE 10.—Male, sixty-five years of age. Disease—coronary thrombosis; myocarditis (chronic). Previous symptoms—*embolic infarcts* (source of embolus not indicated): Pulmonary embolism, lower right lobe; cardiac infarct and aneurism of left ventricle; pleurisy with effusion.

CASE 11.—Female, forty years of age. Disease—chronic parenchymatous nephritis; myocarditis (chronic). Previous symptoms—*embolic infarcts* (source of embolism not indicated): Emboli in both branches pulmonary artery; infarct—right lower lobe.

CASE 12.—Male, fifty years of age. Disease—cardiac hypertrophy and dilatation; chronic diffuse nephritis. Previous symptoms—*embolic infarcts* (source of embolus not indicated): Thrombus—attached to fibrosed area left ventricle; pulmonary emboli—both branches of artery; infarct and pleurisy lower right lobe; chronic passive congestion of lungs.

CASE 13.—Male, forty-one years of age. Disease—cardiac hypertrophy and dilatation; chronic nephritis (not diffuse). Previous symptoms—*embolic infarcts* (source of embolus not indicated): Pulmonary embolus in branch to infarcted right base; passive congestion of lungs; kidney—infarcts.

CASE 14.—Male, fifteen years of age. Disease—chronic endocarditis. Previous symptoms—*thrombosed peripheral veins:* Thrombosis of left jugular vein and subclavicle vein; marantic multiple pulmonary infarcts.

CASE 15.—Female, seventy-one years of age. Disease—coronary thrombosis; chronic myocarditis. Previous symptoms—*thrombosed peripheral veins:* Atheromatous aorta and attached mural thrombi; occlusion of left coronary artery and main branch of pulmonary artery; fibrosis of wall of left ventricle—attached mural thrombus; multiple pulmonary infarcts; thrombosis of left external jugular and right ovarian.

CASE 16.—Female, thirty-four years of age. Disease—chronic endocarditis; parenchymatous nephritis. Previous symptoms—*marantic infarcts:* Hæmothorax and peritoneum; coronary sclerosis; aneurism of aortic arch compressing pulmonary artery; bilateral pulmonary infarcts; mesenteric infarcts.

CASE 17.—Male, fifty-one years of age. Disease—cardiac aneurism; chronic diffuse nephritis. Previous symptoms—*marantic infarcts:* Left ventricle—aneurism with adherent lamellated clots; pulmonary infarcts—multiple bilateral.

CASE 18.—Female, seventeen years of age. Disease—chronic endocarditis. Previous symptoms—*marantic infarcts:* Hæmorrhagic infarcts of lungs; left empyema.

CASE 19.—Male, fifty-four years of age. Disease—chronic endocarditis. Previous symptoms—*marantic infarcts:* Infarcts—right multiple pulmonary.

CASE 20.—Male, fifty-seven years of age. Disease—cardiac hypertrophy and dilatation; chronic nephritis (not diffuse). Previous symptoms—*marantic infarcts:* Infarcts—multiple pulmonary bases.

CASE 21.—Male, fifty-three years of age. Disease—aortic aneurism; pulmonary infarct "pneumonia". Previous symptoms—*marantic infarcts:* Infarcts—multiple pulmonary bases.

CASE 22.—Female, fifty-three years of age. Disease—chronic and acute endocarditis. Previous symptoms—*marantic infarcts:* Chronic endocarditis—mitral and tricuspid; acute endocarditis—mitral; multiple pulmonary infarcts.

2. Pulmonary infarction is not necessarily embolic. In thirteen cases, the thrombus probably was metastatic, but in five of these, the source was not determined. In eight cases, pulmonary infarction was dependent on local marantic thrombosis, in the presence of chronic passive congestion, and the toxic and infective effects of the underlying disease.

3. The vessels, whose occlusion is associated with pulmonary infarction, tend to be of medium size—in fourteen cases.

4. In the rare instances when peripheral venous thrombosis occurs, it is predominately in the veins of the upper part of the body—two cases. (Of twenty-six cases of peripheral venous thrombosis collected by Welch,[3] seventeen were of the upper part of the body. "When we consider that the ratio of thrombosis of the upper extremity to the lower extremity is one to fifty, the relatively large number of the former associated with cardiac disease is certainly most striking." It may not be irrelevant in this discussion to cite the reason for the predominance of thrombosis in the veins of the upper part of the body on the *left* side,—the obliquity and greater length of the left innominate vein similar to those of the left common iliac vein.)

5. Death can seldom be attributed to the immediate effect of the embolus—one case, No. 6. (In the case of No. 7, above, cardiac embarrassment due to the large cardiac thrombus was probably contributory to death.)

From the clinical records of the above cases, we have not been able to determine any change in the level of temperature, pulse, and respiration caused by the onset of pulmonary infarction.

As would be expected, the pulmonary involvement plays but a minor part in the severity of the condition and is only a slight increment to the existing discomfort of the patient. It is of prognostic value because it reflects the extent of right heart decompensation and not because of its intrinsic significance. The age distribution is as follows:

Age	*-9*	*-19*	*-29*	*-39*	*-49*	*-59*	*-71*
Autopsied cases	—	2	1	2	3	11	3

II. *Unautopsied Cardiovascular Cases.*—Examination of the records of the unautopsied cases, again indicates the marked preëminence of cardiovascular disease in association with pulmonary infarction. Of fifty-six cases, thirty-one are cardiovascular. The age distribution is as follows:

Age	*-10*	*-19*	*-29*	*-39*	*-49*	*-59*	*-69*
Cases	1	5	4	7	4	8	2
Mortality	0	4	2	3	2	7	1

The incidence is more marked in the middle and last thirds of life, but not to such an extent as in the surgical cases. The distribution of cases between the sexes: Male, nineteen; female, twelve.

The higher incidence in males is even more marked in the autopsied cases: Male, sixteen; female, six.

III. *Symptoms.*—The manifestation of symptoms is very variable. In only two cases was there collapse followed by death. The other fatal cases suffered the more gradual death due to cardiac decompensation. The temperature-pulse-respiration reaction depends on the general condition of the patient rather than on the pulmonary infarct. Most of the patients—seventeen—had a constant increased temperature whose level was not interrupted by a significant rise to indicate time of infarction. Indeed, infarction probably is not the sudden process which it is in surgical cases, since the condition is preëminently marantic.

In one case there was peripheral venous thrombosis prior to the pulmonary symptoms. This, of course, does not mean necessarily that the peripheral thrombus was the source of the pulmonary infarct. Two cases suffered from visceral infarctions as well.

Characteristic bloody sputum was present in nineteen of the above series, one being marked also by hæmoptysis. It was absent in eleven cases. In the autopsied cases, bloody sputum was predominately lacking in thirteen, and present in seven. This reversal of the usual frequency may have been due to the moribund condition of the patients.

Frequency of Pulmonary Infarction in Cardiovascular Conditions.—One case was diagnosed as bronchopneumonia. Three cases were not considered as pulmonary infarction, though the symptoms and physical signs as recorded are very suggestive. Of thirty-eight cases of chronic cardiac valvular disease occurring between 1923 and 1925, the majority had an occasional hæmoptysis or other signs of pulmonary congestion. Of forty-five cases occurring between 1915 and 1917, three cases showed frank pulmonary infarction, three cerebral embolism, one phlebitis of the veins of the right leg and also thrombosis of the right posterior tibial artery.

Phlebitis and Pulmonary Infarction.—Two cases of phlebitis were puerperal, and, strictly speaking, do not belong in the medical group.

In these cases, as in the surgical series, phlebitis frequently follows the pulmonary infarction; in five cases, particularly in No. 1 and No. 2, the history of pulmonary symptoms covers a fairly long period and suggests incipient tuberculosis—loss of weight, night sweats, etc. In pulmonary infarction, the localization of signs to the bases may be of differential value. (See Table V.)

TABLE V.

Summary of Ten Cases of Phlebitis and Pulmonary Infarction.

CASE 1.—Female, twenty-two years of age. Presenting symptoms—history like incipient tuberculosis. Pain—mild. Sputum—hæmoptysis. Relation of phlebitis—three days after admission, all extremities. Previous history—six years before, typhoid with phlebitis of both legs; pulmonary infarct, two weeks before tonsillectomy; bilaterial pleural pain. Symptoms of phlebitis and pulmonary infarction—right base—consolidation. Result—lived.

CASE 2.—Male, twenty-six years of age. Presenting symptoms—history like incipient tuberculosis. Pain—right chest and flank. Sputum—present. Relation of phlebitis—six weeks before admission, in right calf. Symptoms of phlebitis and pulmonary infarction—right base—400 c.c. bloody serum. Result—lived.

CASE 3.—Male, twenty-seven years of age. Presenting symptoms—history like incipient tuberculosis. Pain—left chest. Sputum—present. Relation of phlebitis—five days after admission, right leg—from ankle to femoral region. Symptoms of phlebitis and pulmonary infarction—bases flat. Result—lived.

CASE 4.—Female, twenty-one years of age. Presenting symptoms—thrombosis left arm. Relation of phlebitis—pulmonary symptoms; embolism; seventh day of thrombosis. Result—died.

CASE 5.—Male, thirty-seven years of age. Presenting symptoms—varicose vein, malaise, dyspnœa. Pain—gall-bladder region. Relation of phlebitis—ten days after admission, left femoral vein signs of phlebitis. Previous history—typhoid. Symptoms of phlebitis and pulmonary infarction—right axilla slightly dull; leucocytosis, diagnosed; acute cholecystitis. Result—lived.

CASE 6.—Female, sixty-three years of age. Presenting symptoms—103° for two months. Pain—right chest. Sputum—present. Relation of phlebitis—sixteen days after admission, right internal saphenous vein. Symptoms of phlebitis and pulmonary infarction—right base and axilla, impaired resonance. Result—lived.

CASE 7.—Female, thirty years of age. Presenting symptoms—105°. Pain—bilateral axillæ. Sputum—present. Relation of phlebitis—thirty-three days after admission, phlebitis bilateral. Previous history—four years before, right leg thrombosis post-partum. Symptoms of phlebitis and pulmonary infarction—twenty-three days after admission, subcrepitant left base. Result—lived.

CASE 8.—Female, twenty-seven years of age. Presenting symptoms—signs on admission and seventeen days after (vaginal examination)—104°. Pain—left chest. Sputum—hæmoptysis. Previous history—puerperium three weeks before. Symptoms of phlebitis and pulmonary infarction—left base—dull mitral insufficiency. Result—lived.

CASE 9.—Female, twenty-six years of age. Presenting symptoms—history like incipient tuberculosis Sputum—present. Relation to phlebitis—miscarriage; thrombosis of pelvic vagina and inferior vena cava. Symptoms of phlebitis and pulmonary infarction—right base—consolidation; left posterior chest, three pints bloody serum; pretibial œdema. Result—died.

CASE 10.—Female, forty years of age. Presenting symptoms—aortic insufficiency; provocative salvarsan. Pain—left chest. Relation of phlebitis—left axillary and brachial vessels six days before infarction. Symptoms of phlebitis and pulmonary infarction—convulsion. Result—died.

The site of pain may be misleading. In Case No. 5, there was pain in the gall-bladder region, but no sputum. There was also a history suggesting the abdomen, probably due to an infarct in the region of the diaphragm.

In two cases, Nos. 1 and 7, there had been a previous phlebitis, which may have left a residuum to serve as focus for the later symptoms. Here, the pulmonary symptoms preceded the phlebitis. In two cases, there was a previous history of typhoid. These cases indicate the value of a complete history. It is probable that some of the idiopathic cases of pulmonary infarction, to be discussed below, may have some such basis.

Typhoid Fever and Pulmonary Infarction.—Between July 1, 1914, and 1917, the following was the partition of complications of typhoid fever. (See Table VI.) Sixty-four complications. Phlebitis (frequently associated with "tender" toes), twenty-one; respiratory (pulmonary infarction was diagnosed in only one instance), eight; bronchopneumonia (three were more probably pulmonary infarctions), six.

TABLE VI.

Summary of Cases of Typhoid Fever and Pulmonary Infarction.

CASE 1.—Male, thirty-four years of age. Onset in course of typhoid—twenty-third day. Pain—right and left chest. Sputum—present. Relation of phlebitis—twenty-fifth day, tender toes; thirty-second day, left calf. Chest findings—twenty-third day, friction rub; thirtieth day, bloody fluid. Result—lived.

CASE 2.—Female, thirty-seven years of age. Onset in course of typhoid—seventeenth day. Pain—left chest. Sputum—present eighteenth day. Relation of phlebitis—twenty-second day, tender right heel; pain along femoral vein; never any signs. Chest findings—eighteenth day, dull, left chest. Result—lived.

CASE 3.—Female, thirty-eight years. Onset in course of typhoid—thirteenth day, left chest; pulse 72–144; respiration from 20–44; nineteenth day; pain—right chest. Duration one month. Sputum—present seventeenth day. Relation of phlebitis—twenty-first day, pain left leg; thirty-first day, pain right leg; thirty-ninth day, pain lower abdomen. Chest findings—eighteenth day, dry crepitant râles left chest. Result—lived.

CASE 4.—Male, forty-nine years. Symptoms—temperature of 105°. Onset in course of typhoid—eighth day, hospital. Relation of phlebitis—left saphenous vein—typhoid bacilli present in blood. Chest findings—eighth day, slight dulness, right base. Pathological findings—myocardial degeneration, left lung, infarct; left femoral thrombus. Result—died.

CASE 5.—Male, thirty-five years of age. Symptoms—temperature of 101°. Onset in course of typhoid—fifteenth day, hospital to thirty-fourth day. Chest findings—left axilla—friction rub right base, dulness fifteenth to thirty-fourth day. Pathological findings—myocardial degeneration; emboli in pulmonary artery; right lower, infarct. Result—died.

Onset of pulmonary infarction is generally late in the course of the disease. Associated with the pulmonary infarction, there generally is a thrombophlebitis (exception No. 1) which may begin as "tender toes" due to irritation of the nerve endings in the wall of the thrombosed vessels, or

to thrombosis in vessels nourishing the nerves. As in other instances, tenderness along the course of the vein need not be accompanied by swelling of the tissues drained (No. 2). In case No. 3, pain in the lower abdomen might have been considered an intestinal perforation, had not the antecedent history pointed to a diagnosis of thrombosis of an abdominal vessel.

The cause of death in the two fatal cases seems to be attributable to myocardial degeneration and to septicæmia (No. 4).

It may be difficult to differentiate pulmonary infarction from intestinal perforation due to the common factor of shock, as the following case report indicates:

> Fifteenth day of illness, the pulse suddenly became imperceptible. Skin—cold, clammy, and moist. White blood cells 26,000—two days previously, 5,000. Blood pressure 60/30. Temperature rose to 106°. Before she could be operated upon for suspected perforation, the patient died. At autopsy, there was no evidence of perforation or of peritonitis. The only positive change was a localized area of pulmonary congestion as in lobar pneumonia.

Miscellaneous Group.—The rarity of the occurrence of pulmonary infarction in acute rheumatic fever, a disease associated with hyperinosis, is apparent. Both patients were unusually old for the disease. In Case No. 3 the diagnosis was based on the therapeutic test. In the other case there was a lobar pneumonia secondary to pulmonary infarction.

Between July, 1914, and 1927, of 147 complications of acute rheumatic fever, three were "lobar" pneumonia, one phlebitis, and one pulmonary infarction. Examination of forty-two cases in that period showed many with signs of pulmonary congestion but none with the characteristic symptom-complex of pulmonary infarction. This, and some other diseases, emphasizes Aschoff's formula that thrombosis is a function of several variables.

The thrombosis and infarction complicating tuberculosis probably are cachectic.

Case No. 8 (Table VII) is another instance of a pitfall—the onset was abdominal pain, pointing to appendicitis.

TABLE VII.

Miscellaneous

CASE 1.—Female, sixteen years of age. Disease—chorea. Temperature—one week after tonsillectomy, 101°. Pain—present, left. Sputum—present. Symptoms of onset—râles only. Result—died.

CASE 2.—Male, forty-three years. Disease—acute articular rheumatism. Temperature—normal until fifteenth day, then 101–104°—septic; normal on twenty-first day. Sputum—present second day; hæmoptysis. Symptoms of onset—fifteenth day, consolidation left lower lobe; thirty-fifth day, condition cleared. Result—lived.

CASE 3.—Male, sixty-four years of age. Disease—acute articular rheumatism. Phlebitis—ninth day, left arm; right leg. Symptoms of onset—twentieth day, collapse, etc. Result—died.

CASE 4.—Female, eight years of age. Disease—acute bacterial endocarditis. Temperature—103°. Symptoms of onset—five weeks, collapse; two days later, collapse. Result—died.

CASE 5.—Male, seven years. Disease—acute bacterial endocarditis. Symptoms of onset—twenty-third day, collapse. Result—died.

CASE 6.—Female, twenty-nine years of age. Disease—tuberculous pleurisy. Temperature—101°. Pulse—112. Respiration—44. Symptoms of onset—collapse. Pathological findings—thrombus infarct, lower right. Result—died.

CASE 7.—Male, twenty-seven years of age. Disease—tuberculosis, dry pericarditis. Symptoms of onset—bedpan, sudden death. Result—died.

CASE 8.—Male, forty-two years of age. Disease—tuberculosis and syphilis. Pain—abdominal. Sputum—present three days after pain. Symptoms of onset—onset, "append." fine râles. Result—lived.

CASE 9.—Male, twenty-seven years of age. Idiopathic—temperature 100°. Pulse—148. Respiration—48. Pain—present. Sputum—present. Symptoms of onset—collapse. Result—lived.

CASE 10.—Female, forty-eight years of age. Idiopathic—cough, bloody expectoration, one month. Temperature—increased. Symptoms of onset—right base, consolidation, crepitant râles. Pathological findings—infarcts right base. Result—died.

CASE 11.—Male, forty years of age. Idiopathic—cough, bloody expectoration. Temperature—104°. Pulse—108. Respiration—24. Pain—abdomen. Symptoms of onset—bases slightly dull; fine râles. Result—lived.

CASE 12.—Male, thirty-four years of age. Idiopathic—onset as pneumonia; chill; fever. Pain—back; right flank. Symptoms of onset—right axilla; crepitant râles. Result—lived.

CASE 13.—Male, twenty-seven years of age. Idiopathic—onset as pneumonia; chill; fever. Temperature—103°. Pain—epigastrium. Sputum—present three days after, pain. Symptoms of onset—no physical signs. Result—lived.

CASE 14.—Male, seventy years of age. Idiopathic—high blood pressure 250/130. Temperature—99°–101°. Symptoms of onset—cerebral hæmorrhage. Pathological findings—laminated clot in pulmonary artery, source? Result—died.

CASE 15.—Male, twenty-five years of age. Lobar pneumonia. Symptoms of onset—no details. Result—lived.

CASE 16.—Male, thirty-six years of age. Lobar pneumonia. Symptoms of onset—delirium tremens, sudden collapse. Result—died.

CASE 17.—Male, forty-two years of age. Lobar pneumonia. Temperature—100°. Pulse—80. Respiration—28. Symptoms of onset—collapse; loud râles. Pathological findings—pulmonary thrombosis—vein; left iliac vein; thrombosis. Result—died.

CASE 18.—Male, thirty-eight years of age. Lobar pneumonia—pneumococcus septicæmia. Temperature—100°–103°. Pathological findings—lobar pneumonia; left lung; infarct, right lower; septic. Result—died.

CASE 19.—Female, thirty-seven years of age. Disease—hepatic cirrhosis; chronic diffuse nephritis. Temperature—99°. Pulse—100. Respiration—26. Pathological findings—infarct, right lower lobe; atelectatic bases. Result—died.

CASE 20.—Female, thirty-six years of age. Disease—acute myelitis. Pathological findings—infarct, subpleural. Result—died day of admission.

CASE 21.—Male, nineteen years of age. Disease—chronic cardiovalvular disease. Temperature—100°. Pulse—90. Respiration—24. Pain—right shoulder. Phlebitis—left calf phlebitis. Result—died (cerebral embolism).

CASE 22.—Female, twenty years of age. Disease—chronic cardiovalvular disease. Temperature—103°–100°. Pulse—130. Respiration—28, constant. Pain—left shoulder. Miscellaneous symptoms only—consolidation at lower angle left scapula. Result—died.

CASE 23.—Male, thirty-three years of age. Disease—chronic cardiovalvular disease. Temperature—100°. Pulse—100, constant. Respiration—20. Pain—present, precordial. Sputum—present. Miscellaneous symptoms—no physical signs of infarction. Result—improved.

CASE 24.—Female, fifty-seven years of age. Disease—chronic cardiovalvular disease. Temperature—98.2°. Pulse—45. Respiration—20. Pain—present, precordial. Sputum —present. Miscellaneous symptoms—consolidation; subcrepitant and crepitant râles. Result—died.

In idiopathic pulmonary infarcts, the onset may suggest tuberculosis or lobar pneumonia. The former mode has already been discussed. In regard to the latter, our data are inadequate, and, for the sake of completeness, we present a summary of the findings in pulmonary infarction as against lobar pneumonia from Conner's [8] article: "The signs of consolidation are of short duration; there are anomalous, protracted, pleural signs; there may be signs suggesting fluid but exploratory puncture is negative; sputum is not rusty as in pneumonia, but bloody or clotted; the course of the temperature is atypical. In all of our five cases there was bloody sputum. The location of the pain and the physical signs were varied."

Our evidence in pulmonary infarction secondary to lobar pneumonia is inconclusive. One case, diagnosed as lobar pneumonia, was actually a pneumococcus septicæmia, with many local manifestations, including a septic infarct of the right lower lobe. The other case, on autopsy, was found to have a pulmonary venous thrombosis, as well as a thrombosis of the left internal iliac vein. This is the only instance of primary pulmonary venous thrombosis which we have been able to find in our series.

Twenty cases of leukemia, occurring between 1914 and 1927, were examined. In one case of lymphatic leukemia, "phlebitis" of the left calf and pleurisy with effusion appeared practically simultaneously, with no change in temperature, pulse, and respiration.

The association of thrombosis in the diseases involving changed conditions of the blood is thus rare. It seems that stasis and toxic factors are more important.

SUMMARY

An intensive study has been made of pulmonary infarction and embolism on the basis of a very large number of clinical case records, autopsy reports and X-ray findings. It has been difficult to correlate the many interesting facts which have been brought to light.

Thrombosis is a very frequent process which occurs conservatively in surgery and on the border line of the pathological in old age, and relatively infrequently in association with its characteristic train of unfortunate events. The determining factor is not the thrombus itself, but the type of tissue reaction to the thrombus—complete organization by the intima or organization with canalization being least threatening.

Thrombi in the swifter current of large veins are more readily fragmented and so are early associated with embolic pneumonic symptoms, such as occur within the first week post-operatively, and, as our analysis of data shows, presage the sudden symptoms of massive pulmonary embolism.

Thrombosis with resultant embolism cannot be primarily attributed to

differences among standard technics, to the operative position of the patient, or even to infection at the region of operation. In the fifteen autopsied surgical cases of pulmonary embolism we have found the distribution among types of operation to be inconclusive. In this series we have noted the occurrence of slight pulmonary signs and symptoms which we particularly wish to stress because of their significance as a herald of the cataclysmic events.

It has been attempted to evaluate the importance of pulmonary embolism as the cause of sudden death. Cardiac embarrassment appears from the data to be more important than the sudden encroachment on the margin of safety of lung tissue. The symptoms of pulmonary embolism are strikingly similar to those of sudden cardiac death as in coronary occlusion.

Traditionally, emphasis has been put on the importance of an immediate ante-mortem cause for precipitating the embolism. In several instances, the obvious age of the pulmonary embolus indicates the fallacy and we profess skepticism as to any essential significance of straining at stool, sitting up in bed, and so forth.

In younger individuals, pulmonary infarction is more frequent than massive pulmonary embolism. The onset is earlier and the course mild. We attribute this to the better general circulation because of which thrombotic fragments are washed away and the larger, more disastrous thrombi are not enabled to form later in the post-operative course.

In general, the onset of symptoms is accompanied by a sharp rise in temperature, of shorter duration than the symptoms, and unaccompanied by proportionate changes in pulse and respiration.

X-ray findings in pulmonary infarction are necessarily only suggestive, as the early condition is one of hyperæmia followed by local consolidation, and occasionally by pleural involvement before the terminal cicatrization and formation of the wedge-shaped area.

When phlebitis is associated with pulmonary symptoms the prognosis is better, not particularly because of a change in prophylaxis, but due to the more favorable anatomy of the thrombosed vein.

In cardiovascular pulmonary infarction, the cardiac thrombi and local passive congestion associated with heart failure are important. In contrast to surgical pulmonary infarction, this type is not accompanied by significant changes in symptoms.

In the course of typhoid fever, pulmonary infarction occurs late. Occasionally, peripheral phlebitis apparently follows a pulmonary infarction and may be due to the same cause instead of the former causing the latter.

Pulmonary infarction is unimportant in other medical conditions.

CONCLUSIONS

1. Minor degrees of thrombosis, infarction and embolism are of great frequency.

2. Only the grosser forms give clinical manifestations.

3. Many instances are undoubtedly unrecognized.

4. Occasionally other conditions, especially cardiac failure, simulate embolism.

5. The vital factor is the condition of the heart.

6. No age is immune, but the probability of fatal outcome from disease, injury or surgical procedure increases with advancing years.

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DIAPHRAGMATIC HERNIA

REPAIR BY TWO-STAGE OPERATION EMPLOYING PRELIMINARY EXTRA PLEURAL THORACOPLASTY

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During the last two decades a considerable literature has grown up on the subject of diaphragmatic hernia. From the first case that Ambrose Paré is credited with having described in 1610, until 1908, according to Arnsparger,[1] diaphragmatic hernia had not been recognized more than ten times off the necropsy table. As late as 1912 Griffin collected 690 cases of this condition, of which only fifteen had been recognized during life. But the record has been quite different during these last two decades; as witness the Mayo Clinic's report of fifty-one cases since 1908, with twenty-seven operations and twenty-two survivals, while L. B. Morrison[2] reports that between April, 1922, and June, 1924, he made the diagnosis of diaphragmatic hernia thirty times by X-ray.

Previous to that time, Morrison had reported twelve other cases. All his were cases of congenital hernia of a portion of the fundus through the œsophageal hiatus in the diaphragm. The patients varied in age between six and seventy-six years. The youngest one had presented a history of vomiting from early babyhood, and was a little suggestive of pyloric obstruction. Most of his patients were sent in for gastric studies because of symptoms suggestive of peptic ulcer. The most constant history was that of pain or distress just above or anterior to the ensiform cartilage. The symptoms sometimes resembled those of gall-bladder disease. Difficulty in swallowing, regurgitation and vomiting were frequent. X-ray diagnosis frequently was possible only after several examinations. In order to demonstrate the hernia, of course, it is necessary to X-ray the patient at a time when an abdominal viscus is projecting above the diaphragm and maintaining a patency that will allow gas or barium to enter the heriated portion. It is probable that the symptoms in few of these cases reported by Morrison were severe enough to have justified surgical treatment.

There has been some expending of energy upon the classification of these herniæ. A diaphragmatic hernia is usually defined as a protrusion of an abdominal viscus, or abdominal viscera, through the diaphragm into the thoracic cavity. The opposite condition of protrusion of a thoracic viscus into the abdominal cavity would also meet the requirements, but seldom, if ever, occurs, owing to three factors: (1) The greater fixity of the thoracic viscera, (2) the, in general, greater weight of the abdominal viscera, and the ease with which they displace the lighter thoracic viscera, and (3) the negative pressure in the thorax with the consequent tendency to suck the abdominal

viscera up gradually during the respiratory cycle. Woolsey[3] says that diaphragmatic herniæ are best classified as follows:

1. Congenital. (Present at birth.)
   (*a*) False.
   (*b*) True.
2. Acquired. (Employed in the same sense as acquired inguinal.)
   (*a*) False.
   (*b*) True.
3. Traumatic.

According to Harrington,[4] the congenital and acquired herniæ usually occur (*a*) through the hiatus pleuroperitonalis, (*b*) through the dome of the diaphragm, (*c*) through the œsophageal hiatus, (*d*) through the foramen Morgagni, (*e*) through an embryonic fusion point of the diaphragm. The traumatic ones may occur anywhere, especially those due to perforating wounds. Those due to indirect injury usually occur through an embryonic fusion point. But such data and classifications on the bases of site, embryology or etiology, while interesting philosophically, have little significance from the point of view of clinical surgery.

The operative treatment of diaphragmatic injuries apparently dates from 1886, when Ricolfi reported the operative cure of a stab wound. Neumann, in 1888, operated in the first case of diaphragmatic hernia in which the hernial contents did not protrude through the thoracic wall. In 1893 both Marana and Amante successfully repaired stab wounds of the diaphragm. But it has been only within the last two decades, and mostly within the last one, that the real progress has been made in operative treatment.

The approaches employed by the different surgeons have been varied. They have been abdominal, thoracic and combined. For the abdominal route an incision parallel to the costal margin has usually been employed, though an upper left rectus incision is sometimes used. The thoracic approaches have sometimes been made through openings provided by rib flaps, but more frequently by intercostal incisions, sometimes with resection of the two adjacent ribs. The abdomino-thoracic approach has been made, either beginning as an abdominal incision and then extending into the chest, or probably more frequently beginning as a thoracic incision and then extending through the lateral aspect of the diaphragm and the abdominal wall. This latter approach naturally provides the best exposure and the greatest freedom of manipulation. It is probably the best incision for a very large hernia, but for many smaller ones would not be necessary. Each case, of course, is an individual problem. A few years ago, in the repair of inguinal herniæ, one heard the various surgeons saying that they did a Bassini, a Halsted, or a Ferguson operation as the case might be. Now, it is doubtful that any large per cent. of the surgeons could tell off hand the differences between those operations. They know the procedures employed in all, but do not know them as belonging to any particular operation. Their view is a little more inclusive. To

them each inguinal hernia is a problem in plastic repair surgery, and each condition has to be met in the way best for that individual patient. And, in deciding what is best each time, the extent of the hernia and the condition of the tissues are the main factors, though age, social position and employment are factors that also enter. So, though we find Sauerbrüch, Willy Meyer and P. E. Truesdale preferring the thoracic approach, and find Harrington, with his extensive experience and excellent results, preferring the abdominal approach for most cases, we find the sane and thoughtful Carl A. Hedblom summing up the law and the prophets by deciding according to every individual case.

Now, after the matter of incision has been decided upon, there are several other items that have contributed to making the repair of diaphragmatic herniæ more frequently undertaken with success. One of these is the fact that a Sauerbrüch chamber is no longer necessary for chest operations. The ordinary nitrous oxide-oxygen apparatus (such as the Gwathmey), in the hands of a good anæsthetist, will maintain sufficient intra-thoracic pressure and keep the lungs sufficiently expanded to make chest work reasonably safe and satisfactory. The division or exersis of the phrenic nerve (Sauerbrüch, Kreuter,[5] Harrington), usually in the neck but possibly in the chest, will give a paralysis of the diaphragm that will make the operation easier and give better assurance of a firm repair; though it probably increases the chances of post-operative pulmonary complications. Gastric lavage, before the beginning of the operation, and the leaving of the stomach tube in situ until the operation is completed, or nearly so, as advocated by Harrington, has many advantages. It decreases the chances of aspiration pneumonia. It helps the surgeon to identify the cardia and it relieves the stomach of gas, and thus aids in its reduction into the abdominal cavity. When the operation is performed by the abdominal route, the use of the stiff rubber tube, suggested by C. H. Mayo, should frequently be of considerable aid in cases where there is difficulty in reducing the herniated viscera because of the suction exerted on them by the negative pressure of the thorax above the constricting ring of the diaphragmatic opening. C. H. Mayo[6] inserts a stiff rubber tube from the abdomen through the hernial opening into the thorax, thus permitting air to enter the pleural cavity, thereby overcoming the suction by equalizing the pressure in thorax and abdomen, and so facilitating the return of the abdominal viscera to their proper place. The repair proper is usually done with cromicized catgut, but there should also be used a sufficient number of interrupted nonabsorbable sutures of silk or linen to insure the continued apposition of the diaphragmatic tissues for some time after the chromic catgut may have become absorbed.

Our experience with diaphragmatic herniæ in the human is limited to the one case that we are reporting, but the unusual extent of the defect in the diaphragm necessitated the adaptation of an operative procedure that we feel will allow a number of patients to be cured in the future that have been

hopeless in the past. Our patient was a boy of sixteen, whose diaphragm had been ruptured nine years previously, in an automobile accident. The rupture had increased in size, incarceration of abdominal viscera in the thorax had become more frequent, and signs of cardiac decompensation—presumably from pressure—were beginning. With an unusually fine and sane attitude, they laid the thing before me and stated that their son's condition during the last few months had become so much worse that they thought he could live only a short while longer, unless something were done. They felt that any operation would be a serious undertaking, but that if any method could be devised that would give him a reasonable chance to come through alive and be cured, he and they wanted it tried. After examining him, I agreed with them and asked for time to think it over.

FIGS. 1 and 2.—These two X-rays in July, 1923, taken after a barium meal, show the extent of the defect in the diaphragm at that time. In the first plate the stomach may be seen passing into the chest near the mediastinum, while in Fig. 2 the colon is herniated through the lateral portion of the diaphragm near the chest wall.

We considered a number of procedures. We thought of suturing the stomach into the defect, or of suturing it to the abdominal wall, as Harrington reports doing recently in a similar case that had an opening in the diaphragm so large that he could not close it. We also considered trying a fascia lata transplant to fill the defect. Finally the correct idea struck us, however, and surprised us by its simplicity. It was just to do a preliminary extrapleural thoracoplasty, the operation devised by Sauerbrüch and now constantly done for obliterating the cavities of chronic empyema, for certain cases of bronchiectasis and for unilateral tuberculosis with pleural adhesions. Such a procedure, of course, would allow the closure of almost any sized defect in the diaphragm. All one had to do was to decrease the circumference sufficiently, and the area to be covered by the diaphragm would be correspondingly decreased. After that, the repair of the diaphragm itself should be a relatively simple procedure. This was the procedure employed. The only fear we had, practically, was the development of an empyema. We knew

that it would be necessary to do a transpleural operation for the repair, and knew that the chances were that the chest would fill with fluid thereafter. If that fluid should remain sterile, we would be all right, but if that fluid should become infected and an empyema develop, then there would be a condition for which we would have no adequate treatment; for we felt that after nine years of compression the lung would not expand sufficiently to fill the left chest, and that we could not collapse the chest wall sufficiently to bring it down to the lung. Now one or the other of these would probably be necessary in order to cure an empyema. For an empyema thoracis is seldom cured until dead space is obliterated and the visceral and parietal pleura are brought into contact.

We did the operation as we had planned, however, and cured the diaphragmatic hernia. The patient developed a sterile fluid in the left thorax that became purulent shortly after he got a septic sore throat. During the course of some nine months' treatment, though, we cured the empyema thoracis without approximating the visceral and parietal pleura. He still appears to have a dead space in his left chest, but the lung has expanded some. The only treatment he had was the drainage of the chest and alternate daily irrigations with Dakin's solution and neutral acriflavine 1-2000. During this time he went wherever he desired. He was fed unusually well, stayed in the sunshine constantly, and, I fear, annoyed his neighbors somewhat by playing a trombone, under doctor's orders, to expand his lungs. Eleven months after he went to the hospital for his first operation he is cured of his diaphragmatic hernia and of his subsequent empyema. He is well and hearty, and weighs eighteen pounds more than before we started working on him.

Report of case in detail follows:

Case I.—J. C. C., white, male, sixteen years old.

Family History.—Father and mother living and well, as are also one brother, three half-brothers, one sister and one half-sister.

Past History.—Patient had influenza before his injury, in 1918, and since then has had mumps, whooping cough, chicken-pox and measles. Aside from these contagious diseases and the disability incident to the condition here reported, he has been healthy.

Present Illness.—At the age of six years he was run over by an automobile. He was taken to the Watts Hospital, where he remained four days. It was thought at the time that he was bleeding internally, but an operation was not deemed necessary. About two years later, there began to occur phenomena that he referred to as "his stomach's going away." This occurred about once a week and lasted about two hours at a time. During the attacks his intestines seemed to leave his abdomen, which became flatter, he had pain in the chest and abdomen, and became short of breath. Five years after the accident the patient consulted Dr. Waldo Boone, who made a diagnosis of diaphragmatic hernia, and investigated the matter of having the hernia repaired, but without receiving any surgical encouragement. A year later, the patient had an interesting attack of appendicitis. The patient says that the attack began with pains in the lower abdomen followed by nausea and vomiting. Then the pain localized to the right lower quadrant of the abdomen. Dr. L. S. Booker operated upon him for an acute abdomen, made a left upper rectus incision and removed a ruptured appendix from the thoracic cavity, through the abdominal incision and the hole in the diaphragm. The patient made a good recovery, but after that his "stomach began to go away oftener and oftener." The attacks became

more severe and lasted longer. The last time that his stomach and a portion of his intestines became incarcerated in the thorax, the attack lasted nine hours.

Physical Examination.—The patient was a fairly-well nourished, sixteen year-old boy. His face was slightly cyanotic during attacks. At other times it was ruddy. He looked healthy. His general physical examination revealed an essentially normal youth, with the exception of enlarged tonsils, an upper left abdominal scar, and the chest condition. The respiratory excursion on the left side was diminished, the point of maximum impulse of the heart was to the right of the sternum, and breath sounds were absent from the left chest. Instead, frequently, peristalsis was audible.

Wassermann was negative. Urine was normal, except for a faint trace of albumen. The white blood cell count was 6,800, the hæmoglobin 75 per cent., and the red blood cell count 4,800,000.

FIG. 2.—See Fig. 1.

June 25, 1927, X-ray Report—Stereoscopic Examination.—"The stomach and part of the large bowel are located in the left chest. Opening through diaphragm appears very large. The stomach goes through the mediastinum, and the colon out near the chest wall. T. C. Kerns."

First Operation—July 19, 1927.—Extrapleural thoracoplasty—left. Under nitrous-oxide anæsthesia.

A long curved incision was made, beginning at about the level of the eighth dorsal spinous process at the costo-vertebral angle and extending downwards and forwards. The skin, fat and muscles were freed and portions of the five lower ribs—6, 7, 8, 9, 10—were resected subperiosteally and extrapleurally. Approximately six inches of the posterior portions of each of these ribs were removed. A very good collapse of the chest wall was obtained. The skin was closed with interrupted sutures of silkworm gut and a small rubber tissue drain left in for a few days to take care of any ooze or serum. A pad was placed over the wound to obliterate dead space and keep the chest wall pushed in. His convalescence from this procedure was uneventful, except from a slight infection in the wound. On this account he was kept in the hospital until August 13, when he was discharged with the wound healed.

It had been our original intention to proceed with the second operation two weeks after the first, but owing to the wound infection, it was decided better to send him home for a month or two in order to decrease the chances of a pleural infection in the second operation, the incision of which would have to cross the first scar. This was done, and he was given daily sun baths over the scar while at home.

After the thoracoplasty, his "stomach never went away again," he said. Subsequent X-rays, however, did show parts of his stomach and intestines in the chest, but it is

probable that his feeling of relief was due to the fact that the tension on the hernial ring had been relieved, and he consequently did not have an incarceration. The shortness of breath became worse, however, and the dyspnoea was frequently accompanied by a pulse rate above 140.

Second Operation—September 24, 1927.—Repair of Diaphragmatic Hernia. Splenectomy. Nitrous-oxid anæsthesia. Gwathmey apparatus.

A long intercostal incision was made between the regenerated eighth and ninth ribs. The ribs were soft and did not interfere with the operation. The incision during the operation was extended on down into the upper left abdomen, about one and one-half inches, in order to gain better access to the diaphragm. The entire stomach, the spleen, a considerable portion of the colon and numerous coils of the small intestines were found in the left chest cavity. There were numerous adhesions about the hernial ring of the diaphragm, but none to the lung or pleura. The left lung appeared to be entirely collapsed, and showed no sign of expanding during the operation. The opening in the diaphragm extended from about an inch lateral to the œsophageal hiatus, to within about an inch and a half of the costal attachment of the antero-lateral border of the diaphragm. In order to release the adhesions about the diaphragm and reduce the viscera, it was necessary to incise this small antero-lateral intact portion of the diaphragm, and extend our incision into the abdomen. After this we were able to release the adhesions fairly satisfactorily, and to reduce with ease all the abdominal viscera, except the spleen. This organ was attached to the margin and upper surface of the diaphragm so securely, and the veins in its pedicle were so numerous, thin and distended, that we were unable to free it. Consequently, we ligated the pedicle and removed the spleen. The only other possible course would have been to reduce the spleen and with it turn down a portion of the diaphram. This procedure would have exposed us to the too great chance of puncturing some of the varicose splenic veins when we sutured the diaphragm, and since we were working above the diaphragm, and the resultant hæmorrhage would have been concealed below the diaphragm, we decided the better policy would be to remove the spleen. With the stomach and intestines reduced and packed into the abdomen, and the spleen removed, the repair of the diaphragm became simple. We sutured it with a continuous chromic two catgut, overlapping the edges. We then put in another layer of interrupted linen sutures. An endeavor was

FIG. 3.—X-ray showing condition of chest in summer of 1928, eleven months after operation. The diaphragm on the left is seen to be intact. Beneath it is a gas bubble in the colon. Above, the lung is seen to be partially expanded, but does not extend out to chest wall. A comparison of the size of the right and left sides of the diaphragm, in this picture, shows the advantage gained by performing the preliminary extrapleural thoracoplasty.

made to expand the lung with increased pressure from the gas-oxygen machine, but entirely without success. The wound was closed tight in layers, with chromic one and two catgut. Interrupted silkworm gut was used for stay sutures and skin sutures. The patient stood the operation well.

Subsequent Course.—The patient did well for a week following the operation. He accumulated some fluid in the left chest soon after the operation. We aspirated this on the fifth day and obtained a sterile, straw-colored fluid. Two days later, he developed a septic sore throat. His temperature ran 103–104. October 4 we transfused him. October 7 we aspirated his chest again and found a purulent fluid containing pneumococci. The next day we opened the incision enough to insert a stiff rubber tube and establish drainage. He was transfused again on October 15, and November 8. The tonsils continued to give trouble and remained large, inflamed and cryptic, so on November 30 tonsillectomy was performed by Dr. B. W. Fassett, under local anæsthesia. He began to improve. Luckily his appetite was stupendous. He consumed enormous amounts of food, which probably was his salvation. He had formed the habit of heavy and frequent eating before his operation, having found that as long as he could keep his stomach full the weight of the food would prevent, to considerable extent, the tendency for the stomach to creep up into his chest. This habit stood him in good stead, and kept his vitality up. He was discharged from the hospital December 24, after having been up and afebrile for ten days, but with a cavity in his chest that would hold a quart of irrigating fluid without difficulty. X-rays had shown his diaphragm holding well throughout this time. Upon discharge from the hospital he was instructed to stay in the sunshine on good days, to blow a trombone or anything else that would help expand his lung, and to irrigate the chest cavity alternately with Dakin's solution (chlorazene) and neutral acriflavine. He reported back at two weeks' intervals for observation.

FIG. 4.—Picture of the patient eleven months after the first operation. He looks well nourished and healthy. The lower incision was the one used in obtaining the collapse of the chest wall, while the incision beginning beneath the scapula was made at the operation for the actual repair of the hernia. Just above the point where the two incisions cross is seen the site occupied by the drainage tube for eight months.

When he left the hospital, I expected to attempt a complete collapse of the left chest at a later date to cure the empyema, but hoped to get some lung expansion before doing that. We were pleased and surprised to find that the cavity diminished in size much more rapidly than we had even dared to hope. In the meantime, he went where he pleased, played, worked a little and appeared to suffer no inconvenience. The cavity gradually got to the place where it would hold no solution. June 11, 1928, we admitted him to the Rainey Hospital, removed his tube, X-rayed him and observed him for two

weeks. He was allowed to go where he pleased, reporting every four hours, however, to have his temperature taken. His temperature was normal throughout his stay. He was full of pep, looked robust and weighed one hundred and twenty-six pounds, which was eighteen pounds more than he weighed eleven months previously, upon his admission to the Watts Hospital for his first operation. Blood pressure was 96/60. Chest wound well healed. Breath sounds audible in left chest. The P. M. I. of the heart was in the fifth interspace, two and one-half inches from the mid-sternal line. Chest measurements:

Expiration, 28½ inches. Inspiration, 30 inches.

Right side: Expiration, 15¼ inches. Inspiration, 16¾ inches.

Left side: Expiration, 13¼ inches. Inspiration, 13¼ inches.

Thus he had one and one-half inches expansion, all of which occurred on the right side, the measurements being made at the level of the nipples.

October 14, 1928, he was seen again, and examined and found to be in excellent condition. He says that he is now as well as anybody. He weighs 138 lbs.—which is 30 lbs. more than he weighed before our first operation.

CONCLUSIONS

Diaphragmatic herniæ that are too large for repair by other means, may be repaired by a two-stage operation, the first stage of which consists of a preliminary extrapleural thoracoplasty, involving the lower chest on the affected side. The second stage—the repair operation—may be done through either a thoracic, abdominal, or combined thoraco-abdominal incision. The case here reported was done through a thoraco-abdominal incision. The patient received a cure. It is interesting to note that he recovered from a post-operative empyema thoracis without approximating the visceral and parietal pleuræ.

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THE NORMAL THICKNESS OF THE PYLORIC MUSCLE AND THE INFLUENCE ON IT OF ULCER, GASTRO-ENTEROSTOMY AND CARCINOMA*

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When gastro-enterostomy brings relief to a patient with peptic ulcer the result is so prompt that it is hard to conceive what the mechanism can be. One thinks first of a lowering of acidity and lessened irritation to exposed nerve endings, but in that case one would expect from Palmer's observations on healing ulcers to see the relief come more gradually. Furthermore, it would be hard to explain the prompt relief of symptoms often observed in persons who after operation do not get much reduction in acidity, or in persons who have a low acidity to begin with.

It would probably be easier to explain the sudden cessation of pain on the basis of prompt relaxation of the spasm in the pyloric muscle which almost certainly is present in many cases of ulcer.[9] Certainly when obstruction is present and the muscle throughout the pyloric segment is hypertrophied there can be little doubt about the existence of strain and overwork, and little question but that the shunting of the current through a new channel should bring immediate rest to the tissues about the pylorus and the ulcer.

There are reasons for believing also that with the entrance of food into the upper part of the jejunum, reverse ripples will run back up the duodenum to produce relaxation and inhibition of the cap and pyloric ring.[1] Such relaxation and cessation of function might well lead to atrophy of the muscle, and some observations by Truesdale suggest that this actually takes place. He found the pyloric muscle to be hypertrophied in fifteen cases in which the lower end of the stomach was removed for duodenal or gastric ulcer and atrophic in three cases in which resection was carried out some time after gastro-enterostomy. These observations were so few in number that we thought it best to reinvestigate the subject in more detail. To do this we measured the thickness of the pyloric muscle in 165 stomachs removed at necropsy and preserved in Kaiserling solution at The Mayo Clinic.

Technic.—A longitudinal cut was made through the anterior wall of the lower end of the stomach midway between the curvatures, and the pars pylorica, with mucous surface downward, was flattened on a piece of cork so that, with the knife held vertically, sections could be cut longitudinally through the region of the sphincter. In normal stomachs there was so little

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variation in the thickness of these sections that measurement of one or two was sufficient, but when the pylorus was much deformed by previous ulceration it was necessary either to take the average of several measurements made in different parts of the ring or to discard the specimen. Most of the sections so discarded were from stomachs with gastro-enterostomy openings. In them the pylorus was often so twisted and the muscle so divided by scar tissue into thinned and thickened segments that no generalization could be made in regard to its width.

TABLE I

Thickness of the Pyloric Muscle in Normal Subjects

Age, years	Sex	Embalmed	Height, cm.	Weight, kg.	Thickness, mm.	Estimated thickness*
59	M	+	182	86.1	8.2	6.5
64	M	+	182	90.6	7.0	6.6
54	M		167	58.9	4.8	5.3
49	M	+	180	56.6	4.3	5.3
41	M	+	182	81.6	6.2	6.3
52	F	+	159	49.7	4.6	5.0
59	M	+	181	72.5	8.5	5.8
21	M	+	173	65.7	4.8	5.8
80	M	+	170	86.2	6.2	6.5
20	M	+	174	61.2	4.6	5.4
29	F	+	168	59.8	5.6	5.4
75	F		147	74.8	4.4	6.0
63	M	+	177	68.0	4.4	5.7
32	M	+	180	61.2	3.8	5.4
56	M	+	177	61.2	6.9	5.4
72	M		169	68.0	6.0	5.7
67	F		159	68.0	5.7	5.7
69	M		176	81.5	5.2	6.3
28	F	+	145	43.1	6.5	4.7
43	M	+	178	86.2	5.8	6.5
36	M		156	68.0	4.8	5.7
68	M	+	178	95.2	8.4	6.9
73	M		177	72.5	5.8	5.9
61	F	+	169	90.6	6.0	6.7
67	M	+	163	74.8	7.2	6.0
23	F	+	168	72.5	6.0	5.9
36	M		187	90.6	5.0	6.6
55	M	+	157	63.4	5.8	5.5
61	F	+	155	74.8	5.4	6.0
48	M	+	178	63.4	5.0	5.5
54	M	+	170	81.6	7.0	6.2
83	F	+	170	72.5	6.6	5.9
41	M	+	184	83.8	5.6	6.3
55	F		165	77.0	5.6	6.1
61	M		169	54.4	5.2	5.2
60	M	+	167	45.3	5.2	4.8
50	F		166	90.6	6.0	6.7
50	M		175	68.0	6.0	5.7
60	F	+	163	61.2	6.6	5.4
35	M		180	81.6	6.4	6.3
48	F	+	168	74.8	7.4	6.0
23	M	?	179	59.8	6.6	5.4
52	F		168	77.0	6.7	6.1
17	F		160	52.2	4.0	5.1
13	M		157	40.7	4.8	4.6
15	M		166	34.0	4.6	4.3
19	F		170	34.0	5.8	4.3

* According to weight of the subject. See text.

CHILDREN					
7.....	M	+	107	27.2	3.4
6.....	M	+	119	27.2	3.4
6.....	F		116	22.6	4.8
2.....	M	+	95	13.6	3.6
8.....	F	+	118	34.0	4.2
1.....	M		69	9.1	2.6
OVERWEIGHT PERSONS					
48.....	M	+	177	113.4	6.0
63.....	F		170	136.0	5.0

Most of the measurements were taken from the enlarged image of the muscle as it was projected from a microscope onto a screen, but in a few cases they were made directly from the section with the help of a binocular loupe and a pair of dividers. In order to avoid the possibility of mental bias, the measurer, at this stage of the work, kept himself in ignorance of the origin of the specimen in hand.

Normal Thickness.—For normal controls we measured the thickness of the pyloric muscle in fifty-five stomachs in cases in which there had not been any sign of disease in the upper part of the digestive tract. The original figures are given in Table I. Little use was made of the measurements on the five children from two to eight years of age because such ages are not represented in our groups of patients with diseased stomachs. We excluded also from representation in Figure 1 the two cases in which the subjects weighed more than 113 kg. The interesting point about them is that their pyloric rings were not thicker than normal for persons of average weight.

In view of the fact that some of the subjects were young, some old, some short, some tall, some thin, and some fat, we first looked for correlations between thickness of pyloric muscle and age, height, and weight. We expected to find some because Scammon [5, 6] and others have found a relation between the age of the subject and the weight and capacity of the stomach. Actually when we included all the data from children as well as adults we obtained good correlation coefficients. The best, $+ 0.68 \pm 0.05$, was for thickness of pyloric muscle and weight of body; next was $+ 0.60 \pm 0.06$ for thickness of muscle and height; and last was $+ 0.53 \pm 0.06$ for thickness of muscle and age. In later years after growth stops and height and weight are not so closely dependent on age, these correlations are not so marked, and (with our data) the respective coefficients drop to $+ 0.46 \pm 0.08$, $+ 0.28 \pm 0.09$, and $+ 0.36 \pm 0.08$.

Using the method of partial correlation which enables one to calculate the degree of relationship between two variables while another is held constant, we gained the impression that in adults there is little real correlation between height and thickness of the pylorus, and that the fair degree of correlation with weight is dependent partly on the correlation between weight and age.

It was a question in some cases whether to use in our calculations the normal weight of the patient or the often diminished weight at the time of death; arguments might be advanced in favor of either practice but we used the weight of the body at necropsy.

On account of difficulties in cutting the sections perpendicularly to the surfaces of the tissue and in knowing just where to measure, we feared at first that our figures would have little value, but as we worked with them and found how well they were correlated with body weight and age, we put more trust in them. Almost certainly deductions may be drawn when, as in Figure 1, the mode or the whole distribution shifts to right or left. Small differences in the means are not so trustworthy and they could be accepted only after corrections had been made for differences in the composition of the groups. Because the thickness of the pylorus varies with the weight and age of the subject it would be unwise to compare averages in two groups of, let us say, normal and ulcer-bearing persons unless the percentages of thin and fat and young and old were nearly the same in those groups. One would either have to choose a control group with ages and builds comparable to those of the diseased persons or, what is often more feasible, one could correct the mean of the diseased group by multiplying by a computed factor. The arithmetic of the process has been described elsewhere by Alvarez and Zimmermann. In addition to the corrections already mentioned we had to correct for differences due to the embalming of some bodies and not of others. This was important because in twenty-seven normal persons embalmed before necropsy the pylorus averaged 6.0 ± 0.2 mm. in thickness, and in thirteen not embalmed it averaged 5.5 ± 0.1 mm.

More trustworthy and instructive than corrected means, however, are polygons such as are shown in Figure 1. The first ones made, representing the distributions of the actual measurements obtained in the several groups of normal and diseased stomachs, looked much like those in Figure 1. In the forty-seven normal stomachs the mode or most common measurement was 6 mm.; about the same as the mean which was 5.8 ± 0.2 mm. The range was from 3.8 to 8.5 mm.

The simple type of frequency distribution apparently gave fairly trustworthy information, but because variations of 2 mm. or more in pyloric thickness are commonly due to differences in body size and in the mode of fixation of the tissues, it seemed best to express each measurement in terms of its deviation (in millimeters) from the estimated normal. The normal was calculated from a formula, $P.t. = 0.0415 \times W + 2.9$ (derived from our data on embalmed bodies), in which *P.t.* represents thickness of pyloric muscle in millimeters and *W* represents weight of the body in kilograms. When the body had not been embalmed 0.5 mm. was subtracted from the estimate.

Duodenal Ulcer.—When all cases of "duodenal ulcer," open and healed, were put together, the polygon was found to be somewhat bimodal as it would be if there were two types of ulcer, one producing slight atrophy and the other some hypertrophy of the muscle. This division into two groups was found

to be even more marked when the twenty-nine open and chronic ulcers were charted alone. The measurements from cases of healed ulcer were almost all within the limits of normal. One very thick pyloric muscle was found in a case of syphilis and cirrhosis of the liver; the stomach was much dilated but otherwise there was little evidence of obstruction.

It seemed probable that the presence or absence of obstruction would have an important influence on the thickness of the pylorus so the cases were divided into two groups; one with definite or fairly definite pyloric obstruction and the other in which evidence of it could not be found in any part of the record. As we expected, there was more hypertrophy in cases of obstruction, but the difference was not striking, and one of the thinnest pyloric rings seen was obtained from a stomach in which there had been obstruction.

In twenty-two of the cases gastro-enterostomy had been performed from one to eighteen days before death, but this procedure so far as we could see, in the short interval of time, did not have any effect on the muscle, and the distribution polygon of these cases was the same as that of the open ulcers.

Gastric Ulcer.—The next distribution in Figure 1 represents measurements from twenty-three cases of gastric ulcer and combined gastric and duodenal

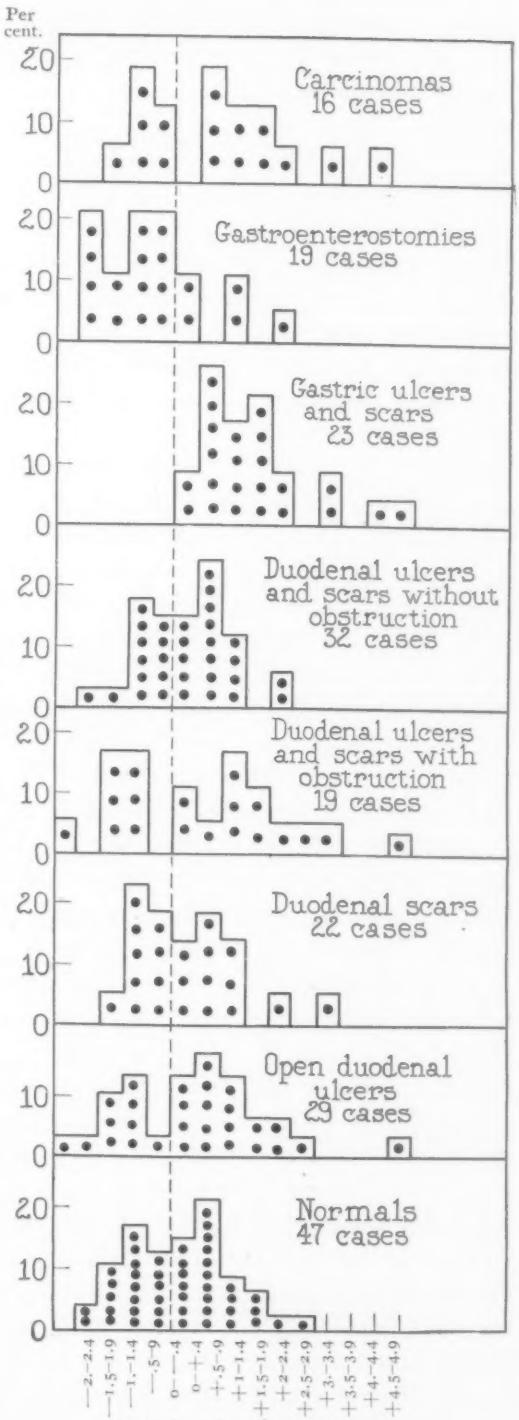

FIG. 1.—Thickness of the pyloric muscle in health and disease. Histograms representing percentage distributions of cases in terms of deviations from the normal mean (estimated according to weight of the subject by formula given in text). The dotted line represents the normal mean.

ulcer. It was hard to know how to classify some of these cases, because in eleven there was ulceration also below the pylorus and in a few the duodenal lesion was the larger and apparently more important one, but we finally decided to put all cases with any gastric ulceration into this group.

It will be seen that in every case the measurements were above the average and in several cases hypertrophy was marked. Apparently, then, there is a decided difference in the way in which the pyloric muscle responds to ulceration above and below the sphincter, a difference due perhaps to the existence of a connective tissue barrier between the stomach and the duodenum.[3] The muscle fibers that make up the sphincter, being more or less continuous with those that surround the pars pylorica, would be more subject to stimulation by ulcers in the lower end of the stomach than by ulcers in the somewhat separate and distinct duodenum.

In twelve cases in which the gastric ulcer or ulcers were situated some distance from the pylorus there were only four with pyloric muscles from 2.0 to 3.2 mm. thicker than normal, and in three of these there was ulceration in the duodenum as well as in the stomach. In eight cases in which the gastric ulcer or ulcers were at or near the pylorus there were two with muscles more than 2 mm. thicker than normal. One, 4.0 mm. thicker than normal, was associated with a very small scar that had produced much narrowing of the pylorus; the other, 4.5 mm. thicker than normal, was associated with an old healed gastric ulcer and a chronic puckering duodenal ulcer. In three cases it was hard to classify the site of the ulcers. Most of the gastric lesions appeared to have been open ulcers. In the nine cases in which obstruction is mentioned or indicated in the record there were two with marked hypertrophy of the muscle, three or four with moderate hypertrophy, and three with muscles within 1 mm. of normal. As has been noted, the two most extreme degrees of hypertrophy were seen in cases of healed ulcers.

Gastro-enterostomy.—The next distribution in Figure 1 represents measurements from nineteen stomachs in which a gastro-enterostomy opening had existed for some time. In one case the interval between operation and necropsy was a month, in another it was two years, in another, three years, and in the remainder it varied between five and seventeen years. In twelve the operation was performed for fairly definite duodenal ulcer and in four for "pyloric" ulcer. In one there probably had been both duodenal and gastric ulcers but the scars were faint; in two there were no scars at all and there were other reasons for suspecting that the gastro-enterostomy had been performed without sufficient cause. In four cases death followed an operative attempt to deal with a gastrojejunocolic fistula, and in one case the patient died of inanition produced by such a fistula. In this case both the duodenal and gastrojejunal ulcers were healed and the pyloric muscle was extremely thin. Another very thin muscle was found in one of the cases in which gastro-enterostomy was performed probably without sufficient cause. Even

in the few instances in which, at necropsy, there was still definite obstruction at the pylorus the muscle was thinner than normal.

In the case in which operation had been performed one month before death the duodenal ulcer, which had given symptoms for fourteen years, was undergoing healing and there was a small acute gastric ulcer near the gastro-enterostomy suture line. The pyloric muscle was 1.7 mm. thinner than the average for a man of 75 kg. In all the other cases the ulcers were healed.

From the appearance of the distribution polygon there would seem to be little doubt that the pyloric muscle tends to become thinned after gastro-enterostomy. In fifteen cases the measurements were below the average.

Carcinoma of the Stomach.—Having in mind the possibility that hypertrophy of the pyloric muscle might be due not only to overwork but to alterations in metabolic rate brought about by adjacent ulceration and inflammation, we studied the effect of carcinoma in the lower end of the stomach. Cases were chosen in which the growth extended almost, but not quite, to the pyloric sphincter. In a few it extended down under the serous coat but care was taken to exclude all those in which an increase in the thickness of the muscle was due to the intrusion of foreign cells.

The last distribution in Figure 1 shows that in two or three cases there was hypertrophy but in the remainder the measurements were within the limits of normal. When the outlet of the stomach is narrowed, made rigid, or blocked by cancer cells one would not expect the strain on the muscle to be so great as it is when a benign, spasm-producing ulcer is present, so it may be that hypertrophy is due sometimes not to overwork but to an abundance of those stimulating tissue-juices described by Burrows and Carrel; juices which may account also for the noncancerous enlargement so often seen in some of the glands situated near carcinomas of the stomach.

COMMENT

The thesis with which we started has received considerable support and it now seems clear that in gastro-enterostomized stomachs, the pyloric muscle tends to be thinner than normal. The fact that in most of those persons with scars in the duodenum the thickness of the pyloric muscle was within the limits of normal suggests that when the muscle atrophies after gastro-enterostomy it does so not simply because the ulcer has healed but because the mechanics of the region are in some way changed.

Strange to say, gastro-enterostomy in infants with congenital pyloric stenosis does not appear to have any effect on the muscular tumor.[4, 10] Perhaps the muscle in these cases is metabolically different from that in the remainder of the stomach so that it can stand out independently much as lipomas do in a person who has lost much body fat.

SUMMARY

In forty-seven adults without gastroduodenal disease the pyloric muscle varied in thickness from 3.8 to 8.5 mm., with an average of 5.8 ± 0.1 mm.

The measurements varied with the weight, height and age of the subject and with the type of fixation (embalming fluid or Kaiserling solution) used.

In the absence of obstruction, duodenal ulcer seems ordinarily to have little influence on the thickness of the pyloric muscle. Occasionally it appears to produce atrophy, and when obstruction is present there sometimes is hypertrophy. Gastric ulcer generally produces hypertrophy.

Gastro-enterostomy tends to produce atrophy of the pyloric muscle. Carcinoma in the pars pylorica ordinarily has little effect, but occasionally it is associated with hypertrophy of the muscle.

These observations lend support to the thesis that the prompt relief of pain after gastro-enterostomy is due at least in part to the immediate removal of strain and overwork from the muscle in the pyloric region.

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PERFORATED PEPTIC ULCER*

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Peptic ulcer is a serious condition, not only because of the suffering and disability it entails, but because of its inherent grave possibilities, such as hæmorrhage and particularly perforation. Emphasis is brought to bear upon this statement by the fact that practically 20 per cent. of ulcers perforate. Inasmuch as there is no way of telling which ulcer will and which will not perforate or bleed, it is no exaggeration to say that every chronic peptic ulcer is potentially a perforated or a bleeding ulcer. Perforation, unless seen early and operated upon early, carries a high mortality. There would be little more for me to say upon this subject than to plead for serious attention to a peptic ulcer syndrome, were it not for the well-known fact that in a certain number of perforating cases the perforation is the first definite indication of the presence of ulcer, just as hæmorrhage, in many instances, is the first indication of ulcer. This, however, is not the rule, in fact fully 90 per cent. of cases have a previous ulcer history, so that the diagnosis of peptic ulcer is of vital importance. The typical history, as outlined by Moynihan about twenty-five years ago, still holds good, although the addition of various tests and such adjuncts as the fluoroscope and the röntgen plate have, to some extent, made for more certainty of diagnosis, especially when the history is vague and not altogether typical. At the same time, it is my experience, and I have no doubt also the experience of many of you, that röntgen-ray diagnosis is not always reliable, that is to say, a negative report cannot be taken as a positive indication of the absence of ulcer. Time and again I operate in such instances to find the most beautiful example of ulcer presenting itself to view. Contrariwise, I occasionally get a report: "deformity of the duodenal cap, indicative of ulcer", and at operation find no ulcer, but a pericholecystitis or frank cholecystitis.

In the perforating case, however, no such uncertainty exists as a rule. That is to say the diagnosis is comparatively easy that an abdominal crisis exists; the crux of the matter lies in early treatment. The value of early surgery has so often been stressed and demonstrated by the favorable figures as far as mortality is concerned, that I need not run the risk of boring you with additional proof. Get them early and you'll get them well, is as valid a slogan today as it ever was. This means, as you well know, getting them before the poison of peritonitis gets a chance to do its deadly work. Nevertheless there are still some who fail to recognize the importance of this and who postpone seeking surgical advice until after the onset of peritonitis, which means, of course, added risk. On the whole, however, I may say that con-

* Read before the Philadelphia Academy of Surgery, January 8, 1929.

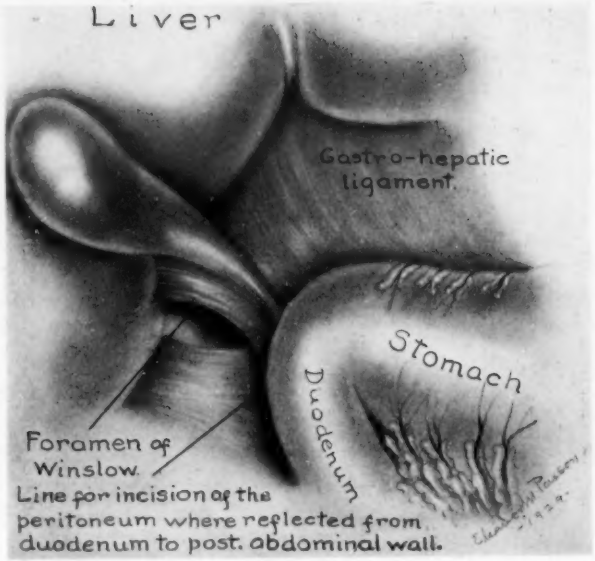

FIG. 1.—Exposure of ulcer on posterior wall of duodenum. Step 1.—Line of section in duodenal-hepatic ligament.

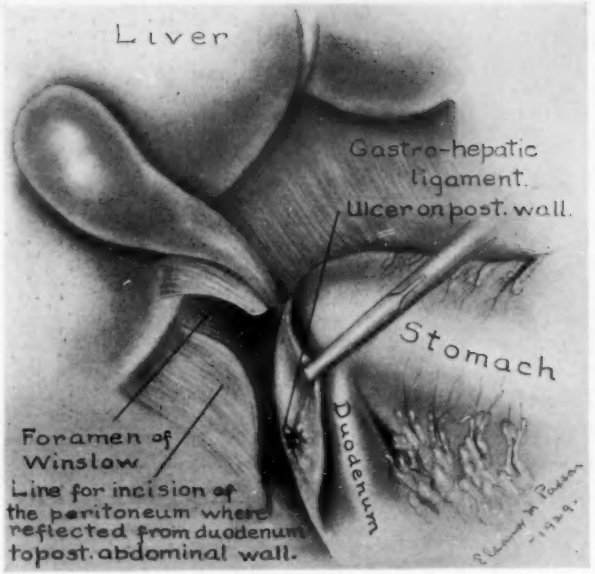

FIG. 2.—Exposure of ulcer on posterior wall of duodenum. Step 2.—Ulcer exposed by rotating duodenum forward.

stant hammering and pleading for early surgery by myself and others like me, who are willing to brave boring the profession by constant repetition, has been effective to some degree. But we must not let up on the propaganda. There are always newcomers in the field who need to be impressed with the importance of this subject.

The onset of perforation is dramatic in its suddenness. It affects the entire abdomen which at once becomes absolutely rigid. The patient is prostrated, although not shocked in the strictly surgical sense of the word. This

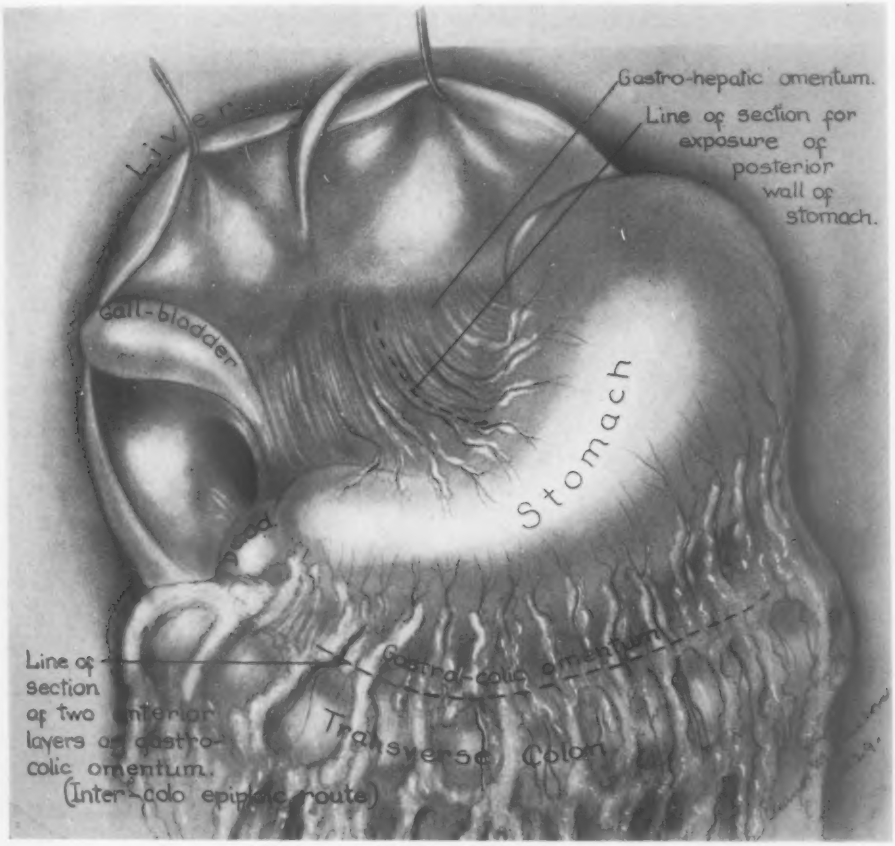
Gastro-hepatic omentum.
Line of section for exposure of posterior wall of stomach.
Liver
Gall-bladder
Stomach
Line of section of two anterior layers of gastro-colic omentum. (Inter-colo epiploic route)
Transverse Colon

FIG. 3.—Exposure of posterior wall of stomach. Step 1.—Line of section.

comes later with the onset of peritonitis, which is avoidable and can be forestalled. One reason why some medical men fail to recognize perforative ulcer is because in the very early stage the pulse rate is practically normal. Nevertheless, the severe prostration together with the excruciating pain, board-like rigidity, and the characteristic short costal respirations as the patient keeps his legs flexed and the body fixed with the hope of easing the pain and protecting the abdomen from examination or contact, these should certainly be sufficient to make the diagnosis. The other signs and symptoms appearing later are vomiting, occasional absence of liver dulness, sometimes

hiccough, shifting abdominal dulness indicative of fluid, followed by cold and clammy skin and finally death, unless the last-named catastrophe is prevented by early operation.

In the face of such a crisis, the thing is to recognize the demand for immediate surgery. This is of much greater importance than to be able to tell whether the crisis is caused by a gastric or a duodenal ulcer, an appendix, a gall-bladder, or a pancreatic hæmorrhage. The abdominal incision will reveal the truth. At the same time there is an advantage and certainly a great satis-

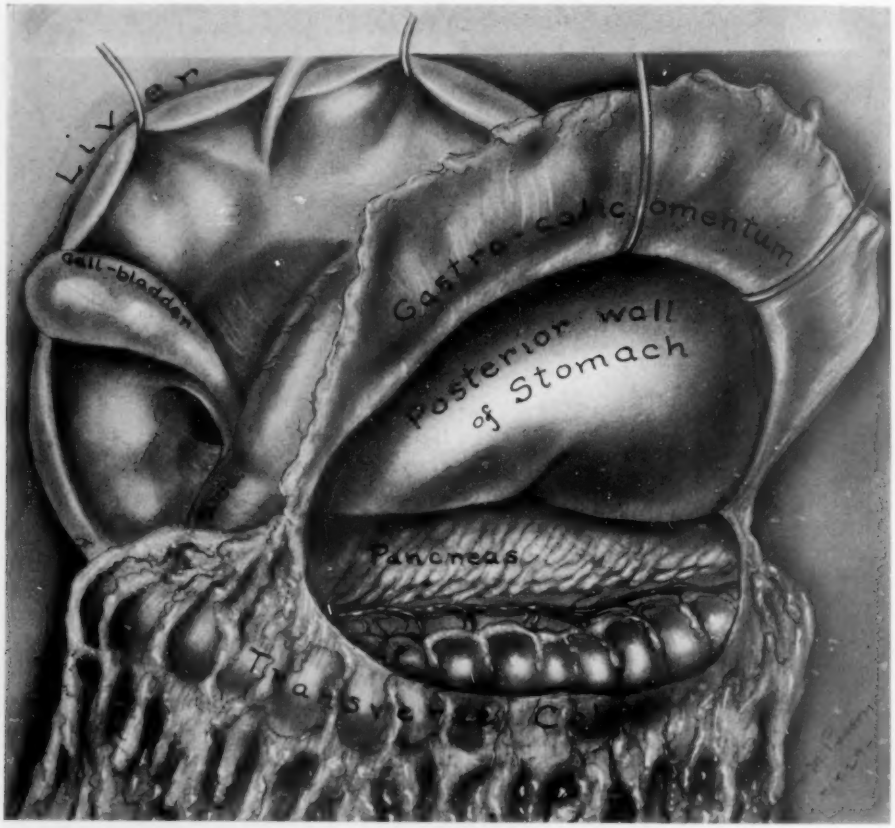

FIG. 4.—Exposure of posterior wall of stomach. Step 2.—Posterior wall of stomach exposed.

faction in being able to differentiate between an acute perforation and an acute pancreatitis. A few words to make this clear may, therefore, be in place. As I have often said, the reason the differentiation is not made more often is the failure to think of the pancreas.

In acute pancreatitis the pain is more excruciating and prostrating than in perforation, and vomiting is repeated, so that intestinal obstruction may be thought of. The difference, however, is that the vomitus consists of gastric or duodenal contents; in addition, the face is livid, and bluish patches of cyanosis may appear over the abdomen and limbs. Halsted was the first to call attention to this cyanosis which he found to occur only in acute pancrea-

titis, although not in every case of that disease—a point to be remembered when examining the abdomen. The rigidity is less marked and is limited to the upper part of the abdomen which is more prominent than elsewhere; tenderness, however, involves the entire abdomen, but is more acute in the upper part and more marked on the left than on the right side.

Once in the hands of the surgeon, what had best be done for the victim of a perforating ulcer? I would say that if the lesion be gastric, I would not, as a rule, deem it wise to excise the ulcer. It not only consumes time but presents the added disadvantage of having a larger opening to close, and entails the risk of dividing large vessels with the attendant danger of hæmorrhage. I prefer to suture the hole through the seromuscular coats, without freshening the edges of the perforation. If, however, this procedure is not feasible, I close the opening either by suturing a tag of omentum over it, or by anchoring the gastrohepatic omentum down to the perforation. If drainage be required, which it seldom is, a piece of rubber dam is placed down to the site of the sutured perforation and a tube is introduced into the pelvis through a stab wound above the pubic bone. In perforated gastric ulcer, in addition to closing the perforation, I make a gastro-enterostomy. In the very late cases where smear and inspection show a virulent peritonitis, I only close the perforation. I always have maintained and still do maintain that a gastro-enterostomy in the absence of peritonitis is a logical and safe procedure, accomplishing both immediate and remote good. Until recently this also was my practice in the treatment of perforated duodenal ulcer. For about two years now, I have been only closing the perforation and in addition have made a wide removal of the anterior half of the pyloric sphincter muscle. Whether the latter can be safely done depends upon the site of the perforation. If the perforation be close to the pylorus, I still make a posterior gastro-enterostomy, except in the very late cases. I find that in the early case the abdominal fluid is sterile, as proven by smears taken and examined as soon as the abdomen is opened, so that there is no contraindication to doing the gastro-enterostomy. It is my practice to smear all acute abdominal cases. The bacteriologist and pathologist are constant companions in the operating theatre. Posterior gastro-enterostomy is particularly indicated where, because of friability of the duodenum, the perforation cannot be satisfactorily closed so that leakage may occur; the anastomosis also relieves tension of the sutures and thus reduces the chance of future ulcer.

The perforated peptic ulcer is occasionally difficult to locate. This applies especially to gastric ulcer on the posterior wall of the stomach in communication with the lesser peritoneal cavity, and to duodenal ulcer on the posterolateral or the posterior wall. An ulcer on the posterior gastric wall that has perforated into the pancreas and has caused a superficial necrosis is more difficult to deal with than the posterior wall ulcer in communication with the lesser peritoneal cavity. Perforated ulcer of the lesser curvature or of the posterior wall close to the lesser curvature is not difficult to handle. The escaping fluid from either is the best indication

of perforation. However, if the perforation be small and there be much exudate that has invaded and bound together contiguous tissues, a careful dissection is required to disclose the walls of the viscus, the site of the pathological opening. To assure a perfect closure, outside of the choice of the anæsthetic, I consider the clear exposure of the site of the leak and of the serosa immediately surrounding it, absolutely necessary for a promising outcome.

Perforated ulcer of the anterior wall of the stomach is very easy to close. Perforated marginal ulcer, if not on the gastro-jejunal margin, can usually be closed without cutting out the anastomosis.

The approach to an ulcer of the lesser curvature or the posterior wall is most satisfactorily made through the lesser or gastrohepatic omentum, while for the ulcer on the posterior wall distant to the lesser curvature and adherent to the pancreas, the approach is best made through the intercolo-epiploic route. This enables one thoroughly to expose the posterior wall. The perforation can then be closed, the necrotic surface of the pancreas cauterized, and a small portion of the great omentum interposed between the affected portion of the pancreas and the site of the closed ulcer. One or more catgut sutures carried superficially through the surface of the pancreas and the omentum will retain the latter.

As I have stated, it was formerly my practice to complete the operation in both duodenal and gastric perforation by a posterior gastro-enterostomy. Recently, however, while I make a posterior gastro-enterostomy in the gastric perforation, I do not, with few exceptions, do so in the duodenal perforation. Making a gastro-enterostomy in perforated duodenal ulcer depends upon the size of the perforation and its distance from the pylorus. Where it is far enough away to allow a generous removal of the entire anterior half of the pyloric sphincter muscle after closure of the perforation, I make the muscle dissection. This dissection is carried half an inch beyond the pylorus on the gastric side and slightly less than this on the duodenal side. The cases I have operated in this manner have made smooth recoveries and are perfectly well, up to the present.

ACUTE INFLAMMATION OF MECKEL'S DIVERTICULUM

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Meckel's diverticula with acute inflammatory changes have been operated upon by the writers four times within a period of seven months (June to December, 1927). An acute abdominal condition so striking and unusual, observed with such frequency, in this brief time, has served to emphasize certain diagnostic criteria by which we were able in the fourth and last case to predict before operation the existence of an acute perforative lesion of Meckel's diverticulum.

The literature abounds with curious instances concerning this congenital structure, its anomalies of position, size, form and histo-pathology, and its relation to other abdominal viscera. The inconsistencies it presents make the pre-operative diagnosis of an involved Meckel's diverticulum perplexing and difficult.

Case I.—A boy, aged eight, was admitted to the United Israel-Zion Hospital, June 19, 1927. Three days prior to admission he had sudden and generalized colicky pains in the abdomen. He vomited several times that day and each day thereafter. The abdominal pains recurred at irregular intervals throughout this period, becoming more marked in the right lower quadrant by the late afternoon of the second day. The temperature then noted was 103° F., falling to 102.4° F. the following day. Under the influence of cathartics there were free bowel evacuations the first and second days, none thereafter.

Physical Examination.—A thin child, with anxious face, evidently very ill and in acute pain, with thighs drawn upon abdomen and legs flexed on thighs. The abdomen was greatly distended and tympanitic. There was some dulness in the right lower quadrant and slight rigidity that was most marked in that area close to the umbilicus. Peritoneal tenderness was present over the lower half of the abdomen, elicited best on the right side just below the umbilical level. Temperature, 99.6° F. Pulse 128. Total number of white blood cells 20,600; polymorphonuclear leucocytes 85 per cent.; lymphocytes 17 per cent.

The pre-operative diagnosis was acute appendicitis.

Operation.—A right McBurney incision. A moderate amount of serosanguineous fluid was found within the peritoneal cavity. The appendix was normal. The ileum in the right lower quadrant was distended and bluish-red. Two feet proximal to the ileocecal area there was disclosed an acutely inflamed Meckel's diverticulum extending across the midline toward the left and upward. It was six inches long, half an inch wide and had no mesentery. There was a small perforation a half inch from the distal end. The tip was intimately attached to a part of the mesentery of the small intestine on the left. Diverticulectomy and stump inversion with peritonealization was the operative procedure. A Penrose drain was placed through the abdominal wound. Recovery took place after a stormy convalescence.

Case II.—A man, aged twenty-nine, entered the United Israel-Zion Hospital, August 28, 1927. During the night of August 26, 1927, he was awakened from sleep by severe pain in the epigastrium which soon became generalized and recurred at varying intervals. The localization of pain was indefinite, but centred mainly about the umbilicus.

Decided nausea was present at times, but there was no vomiting. There was diarrhœa, probably by the use of a cathartic.

When admitted he was suffering moderate abdominal, colicky pains; not acutely ill. The abdomen was tense and much distended. Tenderness was elicited in the right lower quadrant, close to the median line and immediately below the umbilicus. The temperature was 102° F. Total white blood count 9,850; polymorphonuclear leucocytes 84 per cent.; lymphocytes 16 per cent.

The pre-operative diagnosis was acute appendicitis.

Operation and Operative Findings.—Under a right McBurney incision, a long, kinked, but otherwise normal appendix was viewed. Toward the median line was felt a distended loop of small gut, which was pulled into the wound. On this loop and twenty inches from the appendix was a thin-walled diverticulum, two and one-half inches long, distended and bluish-black. The base was as wide as the diameter of the ileal loop from which it was derived (about one and one-quarter inches), the top rounded and lightly attached to a nearby portion of ileum with recent adhesions. This diverticulum had no mesentery. The operation was diverticulectomy, the stump infolded and peritonealized with great care on account of its large size. No drainage.

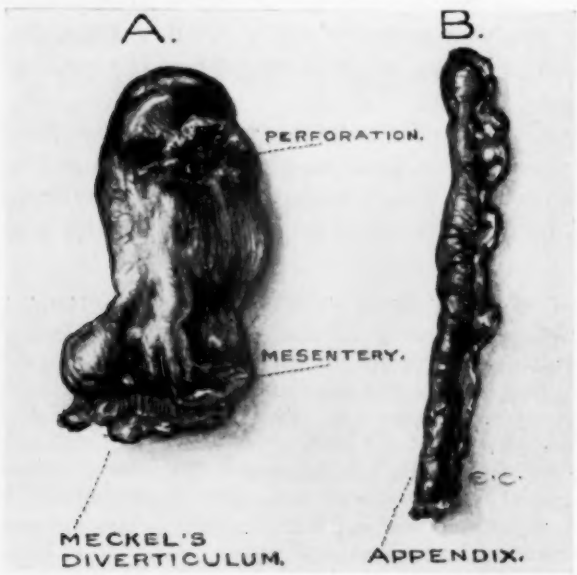

FIG. 1.—Case 3. (A) Meckel's diverticulum, thumb-shaped, with acute inflammation and perforation near the tip. A distinct and thick mesentery is present. (B) Appendix, same patient. (Reproduced black and white.)

Pathological Report.—Acute phlegmonous inflammation of Meckel's diverticulum. Uneventful recovery.

CASE III. A boy, aged nine, was admitted to the Jewish Hospital of Brooklyn, September 11, 1927. During the preceding day he had severe abdominal cramps accompanied by nausea and repeated vomiting. A laxative was taken. Within a few hours the cramps recurred. An enæma was given and a thin watery stool was returned. The temperature was 98.2° F. that evening. The patient had a bowel movement during the night, but its appearance was not noted. Three hours before entrance to the hospital the temperature was 101.2° F., and upon admission had risen to 102.5° F. Pulse 135.

He was a well developed boy with flushed face, acutely ill. There was spasticity and acute tenderness in the right lower quadrant extending toward the midline of the abdomen and close to the umbilicus. Abdominal rebound tenderness was marked. Total white blood count was 20,800; polymorphonuclears 92 per cent.; lymphocytes 8 per cent.

The pre-operative diagnosis was acute appendicitis with diffuse peritonitis, the appendix probably situated near the midline of the abdomen.

Operation.—Through a right McBurney incision, the peritoneal cavity was entered; a grayish gelatinous fluid escaped; located in the right iliac fossa, close to the midline, was an acute gangrenous, perforated Meckel's diverticulum. This was thumb-shaped and unattached, three inches long and one and one-half inches wide at the base, where a thick mesentery with a large blood vessel was noted (Fig. 1 A). There was a large

perforation at its distal third. Diverticulectomy with stump inversion was performed. The appendix, being somewhat thickened, was removed, (Fig. 1 B). One cigaret drain was inserted to the pelvis. Recovery uneventful.

CASE IV.—A boy, aged ten, was admitted to the United Israel-Zion Hospital, November 30, 1927. The acute attack began one day before admission with vomiting and a "stomach ache" around the umbilicus. The vomiting recurred six times that day, but the intense pain decreased in severity. The next day the patient vomited three times. The abdominal pain was moderate and only occasional. After admission, the pain reappeared, but was then most marked in the left upper quadrant. Temperature was 102.2° F. The pulse was 120.

When admitted he seemed acutely ill. The abdomen was distended and slightly rigid. Definite tenderness, especially on rebound, was elicited over the entire abdomen, though less marked in the right lower quadrant. The maximum point of tenderness was one-half inch to the left and immediately below the umbilicus. Total white blood count was 20,200; polymorphonuclear leucocytes 88 per cent.; lymphocytes 12 per cent.

The pre-operative diagnosis was perforated Meckel's diverticulum.

Operation.—A midline and infra-umbilical incision was made. Free pus was evacuated from the peritoneal cavity. Below the umbilicus lay a nipple-shaped Meckel's diverticulum, three-quarters of an inch long, thick-walled, acutely inflamed and with a perforation near the tip. The diverticulum pointed to the left and was about two feet from the ileo-cecal valve. A distinct mesentery had to be ligated before diverticulectomy could be accomplished. The appendix was not disturbed. One rubber drain to the pelvis.

Pathological Report.—Phlegmonous inflammation of Meckel's diverticulum. Recovery uneventful.

Meckel's diverticulum is an occasional sacculation or cecal appendage of the ileum.[1] It is most commonly found in males[2] with an average age of about twenty-two, although it has been noted in infants. In our series of males the ages were eight, nine, ten and twenty-nine.

The incidence of Meckel's diverticulum is low, estimated by Harbin[3] at two per cent. Balfour found only fifteen cases in 10,000 consecutive laparotomies.[4] In 14,000 laparotomies McGlannan[5] encountered three cases. Coleman[6] had 1.8 per cent. in his series. Mitchell, of Chicago, out of 1635 autopsies, reported thirty-nine cases or 2.25 per cent. Adami in all his records gives the incidence as 2.7 per cent.[7] At the Boston City Hospital, in 1,382 autopsies, eleven cases were discovered. Fifteen cases out of 2,600 necropsies were shown at Johns Hopkins Hospital. From 8,133 necropsies at the Dresden City Hospital, only eight cases were recorded.[8]

At the seventh week of fetal development, the mid-gut becomes completely closed off from the umbilical vesicle (yolk sac) by the atrophy of the connecting yolk stalk. The point of final closure of the mid-gut represents a level on the future ileum about two and one-half inches to three feet above the ileo-colic junction. In approximately 2 per cent. of all individuals the intra-abdominal portion of the yolk stalk fails to disappear in a variable degree. This persistent remnant, an intestinal tubular appendage, is known as Meckel's diverticulum.[9]

Meckel's diverticulum is classified as *congenital* and *acquired,* or complete and incomplete. The congenital type is due to a non-obliteration of the fetal vitelline duct. The acquired form results from an original point of

lowered resistance or weakened intestinal wall. This usually occurs along the line of the mesenteric attachment of the intestine, where the vessels pierce the bowel wall and carry therein a sheath of fat. In the complete form of Meckel's diverticulum the sac is formed of all the layers of the intestinal wall. In the incomplete or false diverticula, the sac is formed of mucosa, submucosa and serosa, and it is in reality a hernia mucosæ through a weak point in the muscularis. Though the usual length varies from one-half to four inches, the diverticulum may exist as a nipple-like protrusion or reach a length of thirty-three and one-half inches, as has been reported by Moll.[10] The shape and contour may be tubular, blunt, spherical or pedunculated. The wall is the same as that of the intestine from which it develops. It may contain Lieberkuhn's glands and Peyer's patches. Some writers have reported secreting glands of the gastric type in the distal mucosa, whereas the proximal mucosa contained alkaline secreting glands of intestinal origin.[11]

Ulcer of the duct may occur, and, according to Stulz and Woringer,[12] is due to the presence in the affected diverticula of more or less extensive areas of gastroduodenal mucosa with fundal, pyloric and Brunner's glands. Heteroplasia of the gastric mucosa has been found in about twelve per cent. of cases reported. The histologic picture of diverticular ulcer is that of a gastric or duodenal ulcer or, better still, that of peptic ulcer of jejunum, following gastro-enterostomy (Hubschman). Stulz and Woringer conclude "That the diverticular ulcer is a peptic ulcer of an acute evolution, opening vessels penetrating into neighboring organs, and finally making its way towards the free peritoneum, eventually perforating and producing local or general peritonitis."

The lumen of the duct is usually the same diameter as the ileal gut, but occasionally the outlet is so narrowed as to possess a valve-like action; if entirely closed, a cyst may result. Foreign bodies such as fruit seeds are sometimes found therein.[13] The attachment of the duct is usually opposite the mesentery, but may be at right angles or in its fold. The duct rarely has its own mesentery, though distinct ones were found in two of our cases. The distal end may adhere to any part of the abdominal cavity or neighboring viscera, the most frequent points of attachment being the mesentery, umbilicus, small intestine and abdominal wall. Occasionally, fusion with the mesentery, provides a loop under which the small intestine becomes strangulated. Sometimes it narrows into a fibrous cord which may undergo torsion. The distal end may invaginate into the intestine to produce obstruction; or the duct may remain patent throughout with a resultant fistulous tract leading to the umbilicus (umbilical anus), excreting mucus and occasionally intestinal contents. Meckel's diverticulum may undergo the same pathologic changes as the appendix.

Symptomatology and Diagnosis.—The onset of Meckel's diverticulitis is usually sudden. The clinical course resembles that of an acute inflammatory or perforative lesion of a hollow abdominal viscus, plus partial obstruction of

the bowel. There is no outstanding symptom or sign pointing to a positive diagnosis, but a careful estimation of the more or less constant phenomena may direct attention to Meckel's diverticulum as the possible offending structure. Pain is early, colicky, recurrent and severe. It is generalized about the umbilicus until late changes cause secondary influences in the peritoneal cavity, such as peritonitis, perforation and obstruction. Vomiting is fairly persistent, recurring several times a day. This may be explained by the diverticulum's attachment, recent or old, to nearby structures, thereby involving or ensnaring the intestine to produce partial or complete obstruction. In one of our earlier cases in 1916, a child aged two, the involved diverticulum was attached to the appendix, arching and binding a section of the ileum, so as to cause a complete intestinal block. Distention is prominent and appears fairly early. It is generally widespread, but most evident in the lower half of the abdomen. The marked distention observed in our cases was out of proportion to the tenderness and rigidity, comparing the relative intensity of these signs to other instances of early peritonitis. The localization of tenderness and rigidity in Meckel's diverticulitis is fairly high up in the lower abdomen, usually about one-half inch below the umbilicus to the right or left. Temperature is marked by its high range. A most usual reading is 102° F. to 103° F. The general appearance of these patients indicates a greater toxemia than is usually observed in most early inflammatory lesions: *e.g.* acute appendicitis.

The pre-operative diagnosis of Meckel's diverticulum is rarely made. It was correct in the last of our series of four. Harbin recorded one pre-operative diagnosis out of a series of thirteen. The most frequent abdominal lesion to be differentiated is an acute appendix lying toward the midline, crossing it and extending to the left side. The more frequent pathologic conditions to be considered and excluded are:

1. Perforation of ileum due to foreign bodies. 2. Littré's hernia with obstruction. 3. Involved epiploicae, torsion or inflammation. 4. Diverticulitis of the sigmoid. 5. Acute salpingitis. 6. Primary peritonitis, pneumococcus. The possibility of intussusception must not be overlooked when symptoms of obstruction occur in very young children.

Treatment.—The procedure is diverticulectomy and inversion of the stump with peritonealization. Avoid narrowing and obstructing the lumen of the ileum. When perforation and peritonitis have occurred, the treatment is that of the latter condition. In advanced toxemia or delayed obstruction it is advisable to drain the ileum above the lesion by an enterostomy or by drainage through the base of the resected diverticulum.

SUMMARY

Four patients, males, with Meckel's diverticulitis were operated within seven months; all recovered.

Pre-operative diagnosis was made in one instance based on the repeated or persistent colicky pains centred about the umbilicus, recurrent vomiting,

high temperature, toxicity and distention. The distention was out of proportion to the tenderness and rigidity as observed in acute and early peritonitis. Attention was directed to the signs and symptoms of an inflamed abdominal viscus, plus early and partial obstruction.

In acute abdominal disturbances, when the pre-operative diagnosis is not confirmed at operation, the terminal ileum should be explored for a Meckel's diverticulum.

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THE MORTALITY OF INTESTINAL OBSTRUCTION*

ANALYSIS OF 124 CASES OPERATED UPON AT THE HOSPITAL OF THE UNIVERSITY OF PENNSYLVANIA

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The mortality following operation for intestinal obstruction seems abnormally high. There is a wealth of literature on this subject, but three papers may be quoted to prove the above statement. In 1888 Fitz,[1] of Boston, reviewed the English, French, and German literature from 1880 to 1888 and collected 295 cases, of which 146 were operated on. The surgical mortality was 70 per cent. In 1900 Gibson,[2] of New York, collected 1000 operations with a mortality of 43.2 per cent., a marked improvement, but in 1925 Van Beuren and Smith[3] found 1089 cases published since 1900 giving a mortality of 41.8 per cent., no improvement at all. The great reduction in mortality after 1880 may be explained in part by improved surgical technic and better asepsis, but in part by earlier surgical interference. The conclusion reached by the leading authorities, at the time Fitz presented his paper on intestinal obstruction, was that medical treatment should be tried for at least two days before surgery was resorted to. Previously they had delayed even longer before submitting the patient to operation. It is well known that under such conditions the operative mortality will always be high. The immense amount of research work on intestinal obstruction since 1900 should have led to a still further reduction in the mortality rate.

The experience of Division B of the Surgical Service of the Hospital of the University of Pennsylvania comprises 124 cases operated on from September 30, 1922, to April 1, 1928. There were forty-five deaths, a mortality of 36.3 per cent. As a partial explanation for the high mortality we believe that a grouping of intestinal obstruction is desirable because of the different factors concerned in the etiology, the variations in surgical technics and the different grades of toxæmia encountered. By separating this series into groups we note the following:

Table I.

Intestinal Obstruction.

Division B—September 30, 1922, to April 1, 1928.

	Cases	Recovered	Died	Mortality Per Cent.
Total	124	79	45	36.3
Chronic types	21	17	4	19.0
Acute types	83	58	25	30.1
Acute post-operative ileus	20	4	16	80.0
Total without post-operative ileus	104	75	29	27.8

* Read before the Philadelphia Academy of Surgery, January 8, 1929.

CHRONIC TYPES

In this group are placed the cases in which long-standing subacute or chronic obstruction was evident, but toxæmia absent. There were twenty-one cases classed as chronic obstruction with four deaths. All the deaths were due to post-operative peritonitis. The series included eight cases of cancer of the colon, eight cases of adhesions following previous operations and five cases of miscellaneous lesions. As a whole these patients were advanced in years. The operations were frequently extensive. It seems fair to conclude that the obstruction played a minor part in the mortality of the group, post-operative infection being responsible for the deaths.

ACUTE TYPES

There has been a great deal of experimental investigation directed at acute intestinal obstruction and many attempts to apply this work clinically. This will be discussed later. We were disappointed to find that we failed to lower appreciably the mortality figures from those published in past years. There were eighty-three cases in this group with twenty-five deaths, a mortality of 30.1 per cent. If it were not for the excellent results of the obstructed herniæ series, the mortality figure would be considerably higher.

TABLE II.

Acute Intestinal Obstruction.

Division B—September 30, 1922, to April 1, 1928.

Etiology	Cases	Recovered	Died	Mortality Per Cent.
Obstructed herniæ	44	39	5	11.2
Old post-operative adhesions	14	8	6	42.9
Carcinoma of bowel	13	8	5	38.5
Congenital bands	2	0	2	100.0
Adhesions to mesenteric nodes	2	0	2	100.0
Volvulus	3	1	2	66.7
Tuberculous peritonitis	1	1	0	00.0
Impacted foreign bodies	2	1	1	50.0
Carcinoma of jejunum	1	0	1	100.0
Carcinomatosis	1	0	1	100.0
Total	83	58	25	30.1

From the above table it is evident that these cases fall into four general classes according to the etiology of the obstruction: those due to (1) obstructed herniæ, (2) bands and adhesions, (3) malignancies, and (4) miscellaneous conditions. The surgical problem in each class is quite different, as is also the ease of diagnosis.

Obstructed Herniæ.—From the standpoint of diagnosis and operative technic this group presents few difficulties. The mortality should be practically nil and yet in forty-four cases we had five deaths, a mortality of 11.3 per cent.

No attempt has been made by us to separate the so-called incarcerated from the strangulated group. Filing diagnosis of the inguinal group states that fourteen were incarcerated and six strangulated, and of the femoral

five were incarcerated and seven strangulated, but it seems to us that the border-line cases are judged by the surgeon one way or the other, largely dependent on the color of the bowel. Perhaps this is as perfect a method as is possible, but accurate deductions cannot be drawn. In this series the mortality of the "strangulated" group was 15.8 per cent. and in the "incarcerated" it was 8 per cent.

TABLE III.

Intestinal Obstruction.

Division B—September 30, 1922, to April 1, 1928.

Obstructed Hernia.

	Cases	Died	Mortality Per Cent.
Inguinal	20	4	20.0
Femoral	12	1	8.3
Incisional	7	0	0.0
Umbilical	4	0	0.0
Internal	1	0	0.0
Total	44	5	11.3

Apparently, the fate of the patient depends mostly on the speed with which he is operated on after obstruction (incarceration, etc.) is established. In thirty-nine cases in this group, where the elapsed time between beginning of symptoms to operation was given, it averaged 1.3 days. In twenty-nine cases the time was less than twenty-four hours and only one fatality (3.5 per cent.), in fifteen cases it was less than twelve hours, and in six less than six hours. Of fifteen cases obstructed over twenty-four hours four died (26.7 per cent.).

In 1913 Alexander [4] reported a series of 105 cases of strangulated hernia with thirty-two deaths (30.5 per cent.), ninety-nine were operated on with twenty-six deaths (25.2 per cent.). Thirty-four of these patients were operated on within twelve hours of onset with a mortality of 5 per cent. and twenty-nine were seen after twenty-four hours from onset with a mortality of 62 per cent.

Post-operative Adhesions.—In common with most observers, we found that appendicectomy with drainage and pelvic operations were the most common precursors of post-operative adhesions causing intestinal obstruction. The obstruction occurred from two weeks to twelve years after the original operation. This group carried a high mortality and yet we seem to be helpless in trying to prevent the complication, especially in the drained abdomen. Low-grade inflammation and raw peritoneal surfaces are probably the chief factors causing adhesions in the abdomen closed without drainage.

Diagnosis in this type of case is not always easy and the patient is apt to enter the hospital in a very toxic state. The surgical procedure to be followed requires experience and skill. In some cases the division of a band, thus freeing the obstructed bowel, is sufficient. In others, gangrene of the loop may indicate resection, or, as advised by some, the performance of a technic similar to the Mikulicz's operation. But, at times, in very toxic

patients, the question may arise as to whether it is not better simply to perform an enterostomy.

Carcinoma of Colon.—Unless the patient has been obstructed for a number of days the mortality from this type of obstruction should not be high. The low situation of the obstruction and the fact that dehydration is not a marked factor are important. The technical difficulty of performing colostomy in cases with great distention of the bowel, without soiling, is responsible for the deaths from peritonitis. It seems needless to remark that in the presence of acute obstruction no attempt at radical excision should be made, and, we believe, that in tight distention, without knowledge of the location of the lesion, a cecostomy without exploration should be the primary operation. All of our fatal cases except one died following simple colostomy or cecostomy. In only one case did we attempt a Mikulicz's procedure and this patient lived.

Enterostomy.—Enterostomy and jejunostomy are highly recommended in the literature [5] as life-saving procedures when indicated. Van Beuren and Smith have pointed out the high mortality in the cases of obstruction requiring enterostomy. This procedure is performed usually in the worst type of cases, in one that operation has been too long delayed, and is admittedly the last resort. Under such circumstances it has undoubtedly saved many lives. A patient with a high intestinal fistula, however, must be carefully watched since the fistula itself may be a factor in hastening death. Haden and Orr [6, 7] have shown that, in dogs at least, a jejunostomy may be harmful and not beneficial. The mortality of the cases of acute obstruction in this series which we thought required enterostomy was 60 per cent. This figure agrees with the results of the survey reported by Van Beuren and Smith.[8] It is generally granted that the high mortality following enterostomy is due to late surgical interference, and experimental work has stressed the importance of delay as a factor in causing interference with vascular supply to the obstructed loop.

Delayed Surgery.—The rising mortality of delayed surgery in acute obstruction has been pointed out by most observers. In Finney's [9] series the mortality in the cases operated on in less than twelve hours was 5 per cent.; twelve to twenty-four hours, 11 per cent.; twenty-four to forty-eight hours, 31 per cent. Moore's [10] figures are as follows:

Onset of Symptoms to Operation	Mortality
0 to 24 hours	0
24 to 48 hours	25 per cent.
48 to 72 hours	33 per cent.
72 to hours	65 per cent.

Tuttle [11] gives these results:

Onset of Symptoms to Operation	Mortality
Under 6 hours	0
Under 12 hours	4 per cent.
Under 24 hours	15.4 per cent.
Under 48 hours	18.0 per cent.
Under 3 days	24.3 per cent.

Our experience has been similar to those reported:

TABLE IV.

Acute Intestinal Obstruction.

Division B—September 30, 1922, to April 1, 1928.

Onset of Symptoms to Operation	Cases	Died	Mortality
0 to 12 hours	17	0	0.0
12 to 24 hours	16	2	12.5
24 to 48 hours	18	11	61.1
48 or more hours	24	12	50.0

ACUTE POST-OPERATIVE OBSTRUCTION OR PARALYTIC ILEUS

Following operation for peritonitis, mostly caused by appendicitis, an obstruction may develop either from the paralyzing action of the suppuration itself or from angulation of the lower coils of ileum, adherent to the wall of an abscess or drainage area. Properly speaking, the latter is not true paralytic ileus, but a mechanical obstruction. The group is best termed acute post-operative obstruction. We have not been able to analyze our seven hundred cases of acute appendicitis operated on during these six years, but can recall many cases with obstructive phenomena which recovered without operation. Twenty were operated upon for post-operative ileus and there were sixteen deaths, a mortality of 80 per cent. The group presents many interesting features for discussion because, despite the complicating factor of the peritoneal absorption, it does seem that the mortality is unduly high.

When total obstruction occurs in these cases "it represents the end result of a progressive series of pathological changes dependent primarily upon the severity and extent of the intraperitoneal infection" (Wilensky [12]). In view of the fact that in such an obstruction there is no one occluding point, but rather a general matting together of adjacent adynamic loops, it seems as if the benefit of enterostomy may have been stressed unduly. It is proper to assume that in the cases of peritonitis with obstructive phenomena that recovered without operation, the peritoneal infection was mastered, the fibrinous exudate was absorbed, and the bowel resumed its normal activity. On the other hand, the patient succumbs when the natural resistance fails to control the peritonitis. It is difficult to ascertain in such cases whether the toxæmia of the peritonitis or that of obstruction is the greater factor in causing death. From experimental observation on animals it would seem that the toxins of obstruction are absorbed through damaged bowel mucosa only. Therefore, unless the obstructive phenomena are paramount, we do not believe that a great deal can be expected from enterostomy. At any rate our experience with this procedure in the acute post-operative group has been disappointing. Nineteen cases in this group had an enterostomy and fifteen succumbed, a mortality of 79 per cent. Of these nineteen enterostomies, at least five were jejunostomies with four deaths, and six were ileostomies with four deaths. Deaver [13] and Wilensky [14] do not consider jejunostomy in paralytic ileus satisfactory, and Wilensky states that in their clinic it is performed in but few cases of peritonitis.

Williams[15] suggests the use of *B. welchii* antitoxin in this type of case as well as in acute mechanical ileus. He believes that the toxæmia of intestinal obstruction is due largely to the absorption of *B. welchii* toxin. We have given the gas gangrene antitoxin to a few cases of acute post-operative obstruction, hoping that if we could neutralize the obstruction toxæmia the patient might better be able to overcome the peritonitis. We were able to observe little apparent benefit. However, we have not given it sufficient trial as yet.

ACUTE MECHANICAL ILEUS

According to most observers, in acute mechanical ileus, death is due to the elaboration and absorption of a toxin in the gut. Certain investigators believe that this toxin is elaborated by the damaged mucosa of the obstructed loop and others believe it is due to the proliferation of certain bacterial flora. The most recent proponent of the latter hypothesis is Williams, whose theories have been referred to. Our experience with the use of *B. welchii* antitoxin has been limited and we are not in a position to discuss its efficacy.

The most interesting experimental observations in recent years have been the chemical changes found in the blood in experimental obstruction. It now seems probable that these changes are, to a large extent, due to the loss of sodium and chloride ions in the vomitus, obstructed loop fluid and urine. There has been some confusion in appreciating the significance of these observations due to the fact that the experimental obstruction has been usually of a different type than that commonly found clinically.

The experimental findings discussed may be summarized as follows:

(1) The higher in the intestinal tract the obstruction occurs the more rapidly fatal it is.

(2) Toxic substances apparently are formed in the proximal segment and are rapidly absorbed through a damaged mucosa, but not very readily through a normal mucosa.

(3) Acute intestinal obstruction should be divided into two classes: those without gangrene (simple), and those with gangrene (strangulated). The latter is much more serious due to toxic absorption through the mucosa.

(4) In *high (simple) obstruction* there is rapid fall in blood chlorides with a rise in carbon-dioxide combining power of the blood and a coincident rise in the non-protein nitrogen and urea.

These changes in the blood electrolytes have been most completely worked out by Haden and Orr. The demonstration by them, that the lives of the animals were greatly prolonged by the introduction of hypertonic saline under the skin and intravenously, led us and probably others to believe that we should be able to lower our mortality in acute intestinal obstruction.

Analysis of our figures by year failed to disclose any improvement. In contrast to this disappointing finding Coleman[16] reports two small series of cases, one in which he did not use hypertonic saline with a mortality of 50 per cent., and the other in which he used three-per cent. salt solution with a mortality of only 11.1 per cent. He credits the improvement wholly to the use of hypertonic saline. His two series of cases follow:

I.

Cases 20—Died 10—Mortality 50 Per Cent.

	Cases	Died
Paralytic ileus	8	4
Intussusception	3	0
Strangulated hernia	4	2
Carcinoma of colon	3	
cæcum		1
sigmoid		1
rectum		0
Volvulus	1	1
Band	1	1

II.

Cases 18—Died 2—Mortality 11.1 Per Cent.

		Cases	Died
Carcinoma of bowel		6	
rectum	3		0
colon	2		0
sigmoid	1		1
Strangulated hernia		4	0
Intussusception		2	1
Band		4	0
Paralytic ileus		2	0

The changes in the blood chemistry described by Haden and Orr were in *high, simple obstruction.* Reviewing Coleman's cases and those in our series we find very few that we can class as such. These changes do not take place in *low, simple obstruction.* In an unfinished piece of investigation undertaken some time ago in the Department of Research Surgery we caused a low, complete obstruction in three dogs. This was done by bisecting the lower ileum and inverting the ends. The changes in the blood electrolytes were noted daily by Doctor Sunderman, of the Department of Research Medicine. There were no consistent marked changes in the non-protein nitrogen, chlorides, or carbon-dioxide combining power of the blood. The maximum non-protein nitrogen was 44, 36, and 28 milligrams per 100 cubic centimetres of blood, respectively. The carbon-dioxide combining power fell about the same as in control anæsthetization. The blood chlorides fell to 579, 567, and 556 milligrams per 100 cubic centimetres of blood, respectively.

Further, an animal with any form of *intestinal strangulation* is not saved by sodium chloride solution. The toxæmia is not relieved by such measures in contrast to the marked improvement seen in animals with *high, simple obstruction* (Gatch, *et al.*[17]).

In the light of the above facts, reëxamining the series of Coleman's cases as given, it seems doubtful whether the administration of hypertonic saline to restore the supposed deficient electrolytes of the blood was the prime factor in the improvement of his mortality rate.

In our series, complete blood studies were done in comparatively few cases. The disturbance of the serum electrolytes as described by Haden and Orr was found only once. In a few cases of duodenal and pyloric obstruction and "vicious circle" following gastro-enterostomy, not included in this

series, the blood did show these changes. We were unable to demonstrate any marked advantage in the use of hypertonic saline over isotonic saline in our acute obstruction series, but we do believe that the parenteral administration of large quantities of isotonic saline is of great value both pre-operatively and post-operatively. This supplies both the electrolytes if needed and the fluid in its most available form to combat the dehydration which is always present.

CONCLUSIONS

1. High, simple obstruction is not commonly encountered in the usual cases classed as acute intestinal obstruction. It is present in gastric, pyloric or duodenal obstruction due to tumor or ulcer and, occasionally, a band across the duodenum or high jejunum.

2. The marked improvement seen in animals with experimental high, simple obstruction, after the parenteral administration of hypertonic saline, cannot be expected in the ordinary case of acute intestinal obstruction.

3. One should not delay too long attacking the primary lesion in attempting to correct the disturbance of the blood electrolytes.

4. Isotonic saline may be preferable to hypertonic saline unless we know the blood chlorides are reduced, since dehydration is always present and we can administer a great quantity of fluid.

5. The most important factor in the high mortality of acute intestinal obstruction is still, what it always has been, delayed operation.

Dr. Selling Brill wishes to thank Dr. George P. Muller for his help and assistance.

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BILATERAL ADENOCARCINOMA OF THE BREAST *

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Carcinoma in both breasts is classified by Benassy in three groups: (1) Carcinoma of a second breast developing after amputation of the first, (2) carcinoma developing in a second breast from an original focus in the first, and (3) primary bilateral carcinoma. Cases in the first group are not uncommon; cases in the third group are rare. In the present case we are confronted also with the question whether the neoplastic disease arose from a single focus [3] in each breast or from multiple foci.

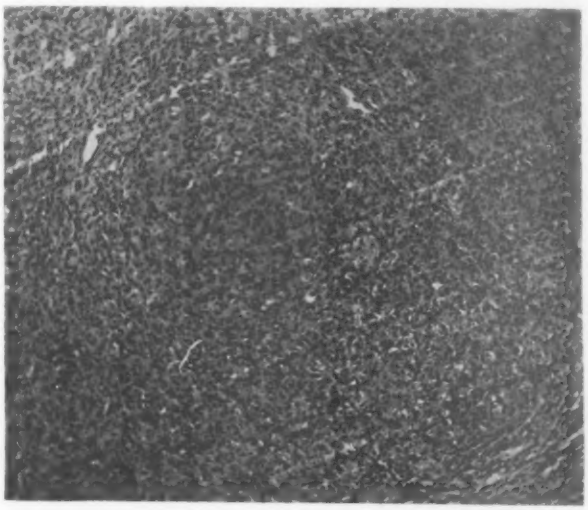

Fig. 1.—Adenocarcinoma of the left breast, graded 4, illustrating the histologic character of the malignant cells (× 100).

A woman, aged thirty-five years, mother of four living, breast-fed children, came to the clinic in January, 1927, with a diagnosis of adenocarcinoma of the left breast. One of her uncles had died of carcinoma, but her parents, four brothers, and three sisters were alive and well. One sister had died of intestinal obstruction. Four months before she came to the clinic the patient had severely bruised her left breast and at that time a nodule, approximately 3 x 2 x 2 centimetres, had been observed. It persisted for three months, was painless, freely movable and did not increase in size. Three months after it first was noted, the nodule was excised by the patient's physician and a pathologist to whom it was sent diagnosed the lesion as adenocarcinoma.

At the time of the first examination at the clinic, the patient apparently was in excellent health. She had gained steadily in weight for several years. Laboratory tests of the urine and blood did not reveal evidence of disease. Röntgenograms of the chest did not give evidence of a pathologic condition. There was a scar on the left breast from a recent operation. Radical amputation of the left breast and axillary lymph nodes was performed. Scattered throughout this breast were eleven nodules of adenocarcinoma varying in size from three millimetres to one centimetre in their greatest dimensions. According to Broders's system, this was an adenocarcinoma, graded 4. (Figs. 1 and 2.) The carcinomatous nodules were distributed fairly regularly throughout the breast. Carcinomatous metastasis could not be found in the left axillary lymph nodes which were

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removed at operation. Histologically, the neoplasm was characterized by malignant, apparently rapidly growing, hyperchromatic cells, which were arranged in ill-defined acini and displayed very slight differentiation. In some areas the cells were closely packed together; in other areas their structure apparently was not so dense.

For ten months after the first operation the patient was in good health. Then she noticed a small "lump" in her right breast below the nipple. This nodule was freely movable, painless and was "watched" for a year by the patient's physician, without apparent change in size or consistence.

When the patient was examined at the clinic the second time she had lost ten pounds. Two freely movable nodules were present in the breast, one at about the five o'clock position and one at about the eleven o'clock position. The röntgenograms of the chest did not reveal any pathologic process of significance. Because of the previous history of carcinoma of the left breast, simple amputation of the right breast was advised and it was decided that this was to be followed immediately by radical amputation if the tumors were proved to be malignant by microscopic examination. After its removal the breast was found to contain thirty-three distinct, light brown to gray nodules; the largest of them was 3 x 2 x 1.5 centimetres in its greatest dimensions and the smallest, 8 x 3 x 3 millimetres. On microscopic examination, all nodules proved to be adenocarcinoma, graded 4. Radical amputation was completed at this time. All of the axillary lymph nodes that were removed were carefully examined grossly and microscopically and evidence of carcinoma was not found in them.

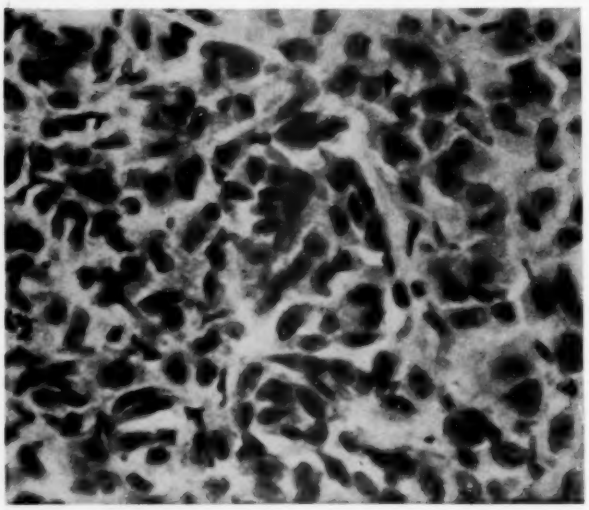

FIG. 2.—Higher magnification of the same area as that shown in Fig. 1, showing more intimate details of the cancer cells (x 350).

Microscopic examination of all nodules in the breast revealed several features. These nodules all closely resembled the carcinoma found in the opposite breast. Generally, the malignant cells were closely packed together in ill defined acini. The nuclei were large and hyperchromatic and some of the cells were multinucleated. All of the nodules were so nearly alike that the sections might well have come from one nodule rather than from the thirty-three. Convalescence was uneventful.

COMMENT

From the standpoint of surgical diagnosis these tumors of the breast are a marked exception to the general rule: although the nodules were carcinoma, graded 4, they were always freely movable, were not hard, and apparently did not increase in size, although observed over a period of several months.

It, furthermore, was unusual that although the tumors in each breast were highly malignant and although they apparently had been present for some time, axillary metastasis was not found. According to Harrington's recent study, the presence or absence of axillary metastasis is one of the most important factors influencing the prognosis in carcinoma of the breast.

From the data at hand it does not seem possible definitely to decide whether these carcinomatous nodules in each breast arose from a single focus in the left breast (the nodule first noted) or from multiple foci of carcinoma in each breast. In the light of Handley's centrifugal permeation studies it would seem that the small nodules of carcinoma in the breasts should have arranged themselves more or less centrifugally around the original focus of carcinoma in the breast, but they did not. The small areas of carcinoma were scattered promiscuously throughout the breasts. It is assumed that the largest nodule was the oldest one.

In favor of the origin of the carcinoma from a single area are: (1) The observation that the tumors apparently were counterparts of each other, and (2) the observation which has been made many times, that carcinoma of one breast is likely to metastasize to the other. In this case it seems that the carcinoma had a marked tendency either to originate in multiple foci or to spread widely in the tissue of the breast rather than to metastasize to adjacent lymph nodes.

Finally, this case illustrates again the fact that after all it is best to remove nodules of the breast and to examine them microscopically if there is any reasonable doubt as to the diagnosis. In this case, the nodules in the breast were multiple, freely movable and although observed for months did not change in consistence. All of these are fairly certain clinical signs of benign tumors of the breast, and still, this patient had multiple carcinomatous nodules in each breast.

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BONE CHANGES IN GAUCHER'S SPLENOMEGALY*

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Gaucher's disease has been defined as a constitutional disturbance of the albumin, fat and iron metabolism which expresses itself in specific changes in the reticulum cells of the lymphatic-hæmopoietic system. Since Gaucher described the disease in 1882, relatively few authentic instances (about fifty) of this unusual condition have been reported. Though the attention of the profession was called to the presence of bone marrow changes in this disease many years ago, the centre of attraction has very naturally been dominated by the striking metamorphosis which the liver and especially the spleen undergo. Relatively little mention has, however, been made of the presence of clinical and röntgenological evidence of the involvement of the osseous system. It is in the hope of focusing interest on this phase of the disease that the liberty is taken of reporting two cases, one of which has already been placed on record and the other of which is here reported for the first time.

The patient, whose history is here again presented, has been treated in several of the large institutions of this city and has been sporadically under the observation of one of us (M.) since 1924. It was only recently that we learned that in that same year, the study and positive diagnosis of this case by splenic puncture had been made the subject of a communication by Mark S. Reuben.[4] Since the clinical findings there noted correspond with those observed by us, the reader may more properly be referred to that excellent study for a statement of the details of the clinical picture. For the sake of completeness, we shall take the privilege of briefly recapitulating the positive findings and of calling attention to certain facts relating to the osseous system which in the press of consideration of the more prominent features of the disease missed the emphasis to which, we believe, they were entitled.

Sidney F., born in 1907, was the second of five children born of apparently normal parents. A younger brother was reported to have suffered from an enlargement of the spleen which, however, could not be demonstrated. The patient had the usual diseases of childhood. There was a history of frequent epistaxis. At the age of two he suffered from an attack of "acute articular rheumatism". At the age of fifteen, the patient again began to complain of pains in the shoulder and knee on movement, though there was no swelling or redness of the joints. At about this time, he suffered a fracture of the right tibia. In 1924, when seen in our office, he walked with a waddling gait and complained of pain in the right hip. No limitation of motion or bone tenderness could be elicited at this time. In 1925, he complained of pain in the right inguinal region which radiated to the right knee. For several years the patient disappeared from observation until he was again seen in 1927 in the out-patient department of the Hospital for Joint Diseases

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complaining of "rheumatism". This time, however, there were definite clinical signs of bone and joint involvement, as the patient walked with a marked waddle and had to be lifted upon the table for examination.

The patient was definitely under-developed both mentally and physically for his age, though appearance suggested that of a wizened, little, old man. The skin was dry and wrinkled and of a *café-au-lait* color. There was a well-developed bilateral pinguécula formation. The abdomen was immense and was seen to be distended by a huge spleen reaching to the crest of the ilium and by the liver which could be easily palpated several fingers' breadth below the umbilicus. No ascites was present. The genitalia were under-developed and the pubic hair showed an infantile growth. Over the tibiæ, however, there was an unusually profuse growth of coarse hair. There was a moderate increase in the size of the submaxillary, axillary, and inguinal lymph-nodes. There was no limitation in the motions of the spine or upper extremities and no bone tenderness could be demonstrated. The knees and ankles were found to be normal and the pain in the knee was presumed to be due to involvement of the hip-joints. The right hip was found completely immobile, probably due to muscle contraction and fibrous ankylosis. The left hip was found in adduction contracture with only slight movement possible. There was no relative shortening. An X-ray of both hips and subsequently of the entire skeletal framework was ordered and was reported in the following sense:

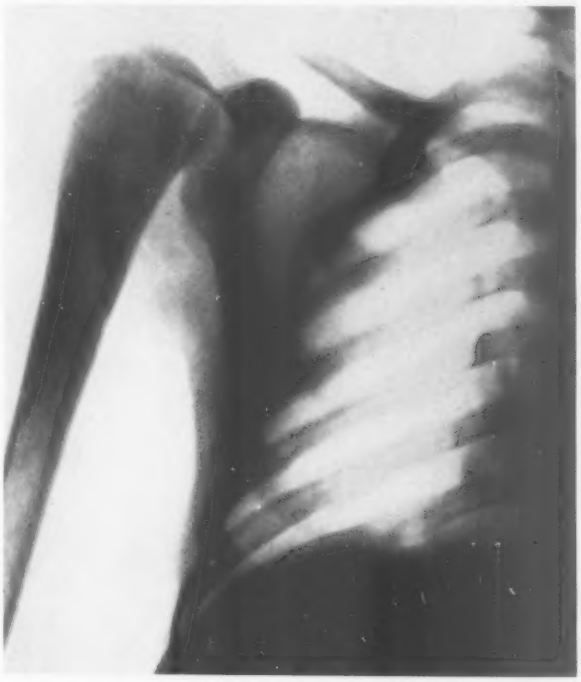

FIG. 1.—Shows irregular absorption of capital epiphysis with mottling of the shaft of the humerus below the epiphyseal line.

"The bones of the skull show definite alterations. The frontal regions show diploëtic absorption with cortical thinning. The bones of the parietal region appear to be normal. The occiput shows definite diploëtic absorption or rarefaction with marked thinning of the cortex. No periosteal proliferation noted. The vascular grooves appear to be slightly accentuated, but there is no definite indication of increased intracranial pressure. The clinoid processes appear atrophic, although the sella turcica is of normal size, shape and appearance. There is no erosion of the floor of the middle cranial fossa.

"The ribs proper show no specific alteration aside from an apparent cortical thinning. The spine shows no changes. Examination of the pelvis shows bone absorption which is more pronounced in the right ilium, particularly about the margins of the acetabulum. Here also there are patchy islands of bone sclerosis. The epiphysis of the right ilium appears to be under-developed, whereas its counterpart of the opposite side appears to be normally formed. The sacrum is negative. Both pubic bones show medullary absorption more marked, however, on the left side, where a small area in the upper anterior angle of this bone appears to have been completely absorbed. The right acetabulum proper

shows slight marginal sclerosis. The left, however, shows an absence of a normal acetabular roof.

"Both shoulder-joints are narrowed and the capital epiphyses show irregular cystic absorption. (Fig. 1.) This change appears to be more marked on the left side. No actual joint destruction is present. The upper ends of the humeri show a mixed process; irregular areas of central medullary rarefaction alternating with medullary sclerosis. The cortex about the areas of rarefaction appears to be thinned out but is not perforated, whereas the cortex in the reactive areas shows definite and fairly pronounced thickening. The middle thirds of the humeri show this medullary rarefaction most characteristically. Examination of the bones of the wrist-joints and hands shows slight atrophy of the epiphysis of the radii with no other demonstrable change. The remaining bones of the hands show medullary expansion without any cortical change aside from slight thinning. The terminal tufts of the phalanges are intact.

FIG. 2.—Shows deformity of the femoral head probably due to replacement of normal bone structure by ingrowth of reticulum cells.

"The right hip-joint shows a patchy sclerosis involving the head and neck as well as the intratrochanteric region. The epiphysis proper is slightly compressed. The neck is foreshortened and there is an early coxa valga deformity present. (Fig. 2.) The left hip shows compression atrophy of the capital epiphysis, the edges of which appear to have undergone moderate proliferation. The femur on this side shows a pronounced medullary absorption throughout. The neck is foreshortened with a tendency toward a coxa vara deformity. The femori show central medullary absorption throughout the upper halves. The middle one-thirds of both femori show medullary and cortical sclerosis. The lower one-thirds, particularly the inner aspects, show irregular bone atrophy or absorption. Some expansion appears to be present in the lower one-thirds of these bones. The left femur shows a slight reactive periostitis along its outer margin, lower one-third. (Fig. 3.) The knee-joints appear to be normal. Examination of both lower extremities shows central medullary rarefaction in the upper one-thirds of the tibiæ with slight thinning of the cortex of these bones. The lower ends, immediately above the ankle-joints, show transverse calcific striations characteristic of an old rachitic process. No evidence of former fracture. Examination of the bones of the feet shows an increase in the width of the medullary cavities, with thinning of the cortices of all the bones of these parts. The tips of the terminal phalanges lack the normal and usual tufted appearance; the terminal phalanges proper being rounded off."

For the details of the second case we are indebted to the courtesy of our colleagues, Dr. Martin Vorhaus, who first saw the case, and Dr. Philip Grausman, who subsequently performed a splenectomy. As in the preceding instance, this patient had been seen in a number of the large institutions of New York City and in one had been subjected to operation for what was believed to be an acute osteomyelitis of the tibia. In this case as in the other the first symptoms were those referable to the osseous system. Though the röntgenographic evidences are by no means as definite as in the first case, there is no doubt in our minds but that this case too must be grouped with those other instances of Gaucher's disease in which the bones have shown involvement.

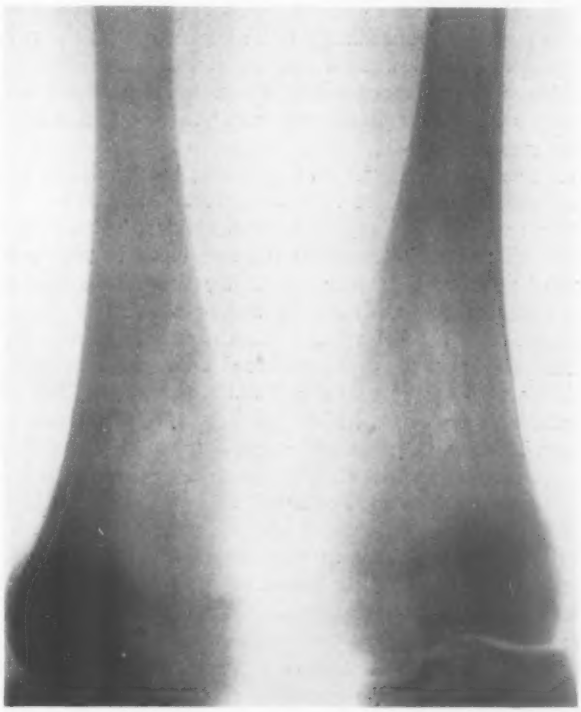

FIG. 3.—Shows expansion of the lower third of the femur associated with medullary absorption and cortical thinning.

Rebecca F., a clerk, aged twenty-one, was the eldest of five children born of apparently normal parents. The four younger brothers showed no symptoms suggesting splenic enlargement. The patient had always been considered "weak". The mother had noticed a marked enlargement of the abdomen since infancy. During childhood, the patient suffered from measles, mumps, diphtheria and pertussis. At the age of one and one-half an abscess on the right side of the neck developed. When five years old, the child fell and thereafter complained of a pain in the right knee and tibia which she attributed to the fall. She was seen at one of the large city hospitals and was referred thence to another institution for the application of a plaster cast. The pain disappeared and the cast was discarded. At the age of nine, she again complained of pain in the right leg and X-ray plates taken at this time demonstrated "something wrong in the right tibia". Again a plaster cast was applied and again after the lapse of several months the pain was relieved. At the age of eleven, because of the presence of a hæmaturia and pains in the right knee, a diagnosis of "rheumatism" was made and tonsillectomy was performed. At this time also the splenomegaly was first noted. For two years following tonsillectomy she was free of symptoms and then as a result of the recurrence of pain, she went to one of the large orthopedic hospitals of this city where a diagnosis of "tuberculosis of the hip" was made and a plaster spica was applied. She was sent to a country convalescent home where she first noticed her "attacks" of headache, nausea and dizziness. The spica remained on for about eight months, when she was examined in another orthopedic institution and absolutely no evidence of hip involvement found.

In 1920, the patient complained of intense pain in the right tibia which rapidly increased in severity so that she was unable to walk. There was no history of chills, no loss of appetite, no malaise except for the pain in the leg. The leg, however, was dusky-hued, swollen and exquisitely tender to touch. A diagnosis of acute osteomyelitis was made and the patient referred to the hospital for immediate operation. The operation was performed by the late Doctor Barrie. "A linear incision was made along the inner anterior aspect of the middle third of the right tibia. An opening through the fat and fascia disclosed a bluish-gray periosteum bulging in all directions. Incision over the periosteum caused spurting of the dark bloody fluid. In all directions, periosteum was stripped from the bone. A wedge one-half inch by one and one-half inches was removed and two small sequestra surrounded by granulations were removed. Visible pus was also present." Immediately following osteotomy, the temperature dropped to normal and remained normal without a single elevation until her discharge to a convalescent home seven weeks after operation. For a period of about two years the bone sinus remained open, occasionally discharging small sequestra, but the pain has been eliminated up to the present time.

During the year or two preceding her admission to the Hospital for Joint Diseases the patient complained of repeated recurrences of her "attacks" of nausea and dizziness. When seen at our hospital the patient presented the characteristic clinical appearance of one afflicted with Gaucher's disease; bilateral pinguecula formation, *café-au-lait* color, small petechial hæmorrhages into the skin of the face and forearms, a liver enlarged to two fingers' breadth below the umbilicus and a spleen reaching to the crest of the ilium, no ascites. The urine and Wassermann tests were normal. The blood picture showed hæmoglobin 60 per cent., red blood cells 3,004,000, white blood cells 4,000, polymorphonuclears 46 per cent., small lymphocytes 40 per cent., large lymphocytes 13 per cent., basophiles 1 per cent., blood platelets 288,000, coagulation time three and one-half minutes, bleeding time one and one-half minutes. Over the inner surface of the right tibia there was an adherent scar about three inches long. At this level the right tibia measured thirteen and one-half inches in circumference as compared with the left, which at the same level measured only twelve and one-eighth inches. There was no pain or bone tenderness either in this region or over any of the other bones. There was no limitation in any of the joints of the body. On March 22, 1927, operation was performed and a spleen weighing 1,332 grams, measuring about twenty-seven centimetres in its greatest diameter and presenting all the gross and microscopic appearances of the typical Gaucher splenomegaly was removed. The patient made an unusually uneventful recovery and since her operation (fifteen months ago) has gained five pounds in weight. Though she still tires easily after exertion she feels a marked increase in her strength. The liver is slightly smaller than before operation and the yellowish color previously present has disappeared. The "attacks" of nausea and vomiting as well as the tendency to hæmorrhage appear to have been completely controlled. The pingueculæ are still present.

X-ray plates taken recently showed a small and rudimentary type of sella turcica. The rest of the bones except the tibia showed no changes. "Examination of the right tibia shows a definite process involving the upper two-thirds of this bone. There is a dense endosteal and periosteal sclerosis with an actual increase in the diameter of the shaft proper. An area of central medullary rarefaction is seen immediately beneath the condyles and may represent active destruction at this point. The lower one-third of this bone shows definite medullary absorption. No periosteal changes are demonstrated over this site. There is a periostitis of the shaft of the fibula at the junction of the middle and upper thirds. The original changes in the upper two-thirds of the tibia cannot, of course, be determined at this time in view of the reactive changes following earlier operative intervention. The process in the lower third of this bone may well simulate the appearance noted in other cases of Gaucher's disease."

Unfortunately, we have been unable to get any of the X-ray plates taken prior to operation by Doctor Barrie. Out of respect for his admittedly great knowledge of the pathological conditions of bone, the possibility of an acute osteomyelitis must be admitted in this case. However, in view of the clinical history of this patient, we feel that this designation may erroneously have been applied to what was in reality a case of Gaucher's disease with bone symptoms. The long history of recurrent, afebrile attacks of bone pain, the sudden onset of the last attack without clinical evidence of a septicemia or even a severe toxemia, the relatively low-grade fever and the abrupt fall to normal temperature following operation, all seem to lend credence to this belief. The "visible pus" seen at operation may well have represented the bone necrosis which Pick has shown is the consequence of the ingrowth of the Gaucher cells. On the other hand, the sudden swelling of the leg, the development of a dusky color to the skin, the exquisite tenderness, the spurting out of "a dark bloody fluid" after incision of the tense, bulging periosteum stripped away from the underlying bone seem to point not to an acute infectious process but rather to a vascular accident, a subperiosteal hæmorrhage, the counterpart of the hæmaturia noted in childhood and the petechial hæmorrhages seen upon admission to our hospital.

It is interesting to call attention to the fact that in these two cases as in two others to be mentioned later, the diagnoses of rheumatism and bone tuberculosis were first made before pathological examination elucidated the true underlying cause. It is further interesting to observe that, though the bone marrow changes were undoubtedly antedated by unnoted enlargement of the liver and spleen, the first symptoms for the treatment of which these patients presented themselves were referable to involvement of the long bones.

The bone changes characteristically seen in this disease appear to have been first noted in 1904 as a result of the clinical acumen of Dr. Emanuel Libman. Nevertheless, in spite of our present knowledge of the bone marrow involvement, the cases in which symptoms indicating such lesions have been recorded appear to be relatively few. Out of a series of nineteen cases which he reviewed, Reuben noted the presence of bone tenderness in only three. Brill, Mandlebaum, and several other authors have recorded bone tenderness en passant in their case histories, but none of these have apparently observed actual bone destruction or change demonstrable either clinically or by röntgenographic examination.

In 1922, Pick[3] called attention to the fact that Gaucher's disease may show marked variations in the extent of involvement of the different parts of the lymphatic hæmopoietic system. In the case he reported the "changes in the spleen and liver were relatively in the background of the clinical-anatomical picture, so that Gaucher's disease, as such unrecognized, gave the impression and received treatment as a bone disease". This patient, a man of forty years, presented no symptoms until the age of twenty, when recurrent pains in the left femur were noticed. Gradually, a swelling of the lower end of the femur and a gibbus in the dorsal spinal region developed. The patient

was treated by traction in recumbency without any relief. Subsequently, he was operated upon for what proved to be a fatal carcinoma at the liver portal, and the autopsy disclosed facts of interest in the present consideration. Apart from the more usual morphological changes in the liver and spleen, which absolutely established the diagnosis, there was extensive involvement of all the bones examined. The pelvic and long bones, especially the femur, showed the presence of huge soft grayish and reddish tumor masses of typical Gaucher's cells in the marrow spaces. The fifth lumbar vertebræ was compressed and wedge shaped. The gibbus, clinically noted, was found to be due to a breaking down and compression of the eleventh and twelfth dorsal vertebræ. In contrast to the findings in tuberculous compression, however, the ligamenta intervertebralia were here found intact, as might probably have been expected in a case of non-infectious origin.

In 1927, Santee,[5] too, reported a case in which the diagnosis was established by splenectomy and in which, as in Pick's case, the first symptoms were referable to the osseous system. The patient, a man of twenty-nine years, was first seen complaining of "a sprain of the left hip, following a jump on a roof, eighteen months beforehand. This was followed gradually by increasing disability in the left hip, particularly on active use". He was treated in a Brooklyn hospital by rest in bed, traction, suspension, walking Thomas calipers, etc., without relief. When seen by Santee, radiograph showed "a slight irregularity and loss of detail lower half margin head of left femur, which is tuberculous in appearance without extensive bone destruction. Head of femur faintly mottled which may be due to slight rarefaction. The presence of a Gaucher's type of splenohepatomegaly was recognized and splenectomy performed. Following operation, the patient's general condition was apparently definitely improved, but six months later, "his hip shows no apparent change".

From the study of the cases which have been noted above, the panorama of practically all of the bone manifestations in Gaucher's disease may be reconstructed. The pathological changes seen in the stage of early bone involvement perhaps have been best described by Brill, Mandlebaum and Libman.[1] "Cells identical with those found in the above organs (liver, spleen, etc.) are found in abundance in the bone marrow. . . . Occasionally a large mass of cells is noted, but for the greater part they are found either singly or in groups of from four to ten cells. In the latter instance the cells are always found in intimate relationship with the walls of the capillaries or attached to the connective tissue reticulum. The larger masses cannot always be seen to have such a relationship, and occasionally they are found surrounded by a considerable number of red blood cells. In a few places the cells are distinctly seen within the lumen of the dilated capillaries."

Unfortunately, the specimen from which this report was written appears to have been taken from the femur of the patient, while the site of the greatest pain and presumably also of the greatest pathological changes was in the tibia. The subsequent opinion of Brill and Mandlebaum[2] that "no charac-

teristic variation in the degree of involvement between the early and advanced cases is apparent" must therefore be taken with a great deal of reserve. In the advanced case referred to before, Pick found extensive disease of the bones with nodular tumor formations as well as diffuse infiltration in the bone marrow. Microscopically all stages of development of the Gaucher's cells were demonstrable in the bone marrow. There was a peculiarly characteristic tendency of these cells to take on long spindle-shaped and fascicular forms. As a consequence of their massive growth in the marrow spaces, the bone trabeculæ gradually underwent atrophy and subsequent disintegration. The end result consisted of a mass of necrotic bone trabeculæ held together by a relatively anuclear fibrous tissue, which developed *pari passu* with the intense proliferation of the reticulum cells.

The clinical correlative of the early pathological change is doubtless envisaged in the vague pains, the "rheumatism" and the bone tenderness already mentioned. As the disease progresses, and as the bone changes become more pronounced, the patient begins to note greater and greater pain and disability in the bones or joints, and the attention of the physician becomes focused more upon the local condition than upon the systemic disease at the basis of the patient's complaints. In the advanced stage, where destruction of the bone has already taken place and deformity has developed, the patient necessarily becomes the object of the orthopedist's solicitude whether the diagnosis has been made or not. Usually, of course, the diagnosis of Gaucher's disease is made by the clinician. Occasionally, as in those cases where symptoms referable to the osseous system appear before the development of the pathognomonic splenohepatomegaly has attracted attention, it may be of value to bear in mind the value of radiologic examination of the affected bones.

Röntgenologically, the changes in the bones in Gaucher's disease are characterized by medullary absorption, cortical thinning and expansion of the bone. The medullary destruction which takes place not as a diffuse process but in the form of scattered islands doubtless represents the sites of proliferation of the Gaucher cells. This alternation of areas of absorption with areas of denser bone formation gives the plate a characteristic mottled appearance. Whether the surrounding areas of relative osteosclerosis represent nature's efforts in reparation or the unaffected internodular spaces, it is impossible for us to say. However, in view of the röntgenologic evidence of bone repair presented by the two cases above reported, it may be presumed that the mottling is due, in part at least, to a stimulation of osteogenic forces. The cortical thinning and the bone expansion are doubtless the result of the increased subcortical pressure exerted by growing cell masses, and are in all likelihood the explanation of the bone tenderness and pains described by the patients. Similarly, the cystic areas of absorption noted in the joint surfaces probably bear witness either to invasion of the cartilage by the cells or to the necrotising effects of pressure of the subjacent cells upon the cartilage. Periosteal changes are usually slight.

Especially in its later stages, Gaucher's disease is usually fairly easily

diagnosed. However, in the type in which symptoms referable to the osseous system predominate, it may occasionally be necessary to exclude the presence of tuberculosis, lues, leukemia or so-called "rheumatism". Clinically, the differential diagnosis may be established by the absence of a leukemia, or a polycythemia, the negative Wassermann, the presence of pingueculæ and the enlargement of the liver and the spleen. Röntgenologically, too, the diagnostic criteria are fairly definite and the differentiation offers no difficulty. Tuberculosis is characterized by the presence of extensive articular destruction and an associated dense synovitis. There is marked atrophy of the bone, but no areas of cystic degeneration in the juxta-articular region as in Gaucher's disease. In this latter disease, the lesions are generalized, while in tuberculosis they are usually localized to one joint. In tuberculosis, in the absence of a mixed infection, there are no periosteal or endosteal sclerotic changes and the mottling described as occurring in Gaucher's disease is not seen. In lues, on the other hand, the marked periosteal and endosteal changes may resemble very closely those seen in Gaucher's disease. However, here there is more evidence of a generalized periosteal reaction with a pronounced tendency to osteosclerosis. Except in cases of superimposed pyogenic infection, lues does not lead to bone atrophy and the characteristic alternation of sclerosis with areas of medullary absorption does not occur. Joint syphilis is usually of a destructive nature as in Charcot's disease, while in Gaucher's disease the articular lesions are more defined by atrophy than by destruction. Recent descriptions of the röntgenographic evidence of bone changes in lymphatic leukemia have stressed the appearance of periosteal reactions. However, these cases do not show the endosteal sclerosis, the atrophy, the joint changes or the medullary absorption which have been noted in Gaucher's splenomegaly.

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OSTEOMYELITIS OF THE VERTEBRÆ

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In previous communications the mechanism and pathogenesis of acute osteomyelitis in general was extensively discussed. The present paper concerns itself with osteomyelitis of the vertebræ of the spinal column. The principles of the pathogenesis of acute osteomyelitis in general will be incorporated in this paper as they apply to osteomyelitis of the vertebræ; and the phenomena and extraordinary features of infection of the latter bones will be found to be caused in the ordinary way and to be explainable by the situation of the lesion and by its relation to neighboring cavities, viscera, or other important structures.

This communication will consider only those cases of acute osteomyelitis of the vertebræ which are caused by the common pyogenic organisms—staphylococci, streptococci, etc. It will not include any cases due to such extraordinary infecting agents as tubercle bacilli, actinomyces, etc.; nor those cases of chronic spondylitis due to typhoid bacilli; nor cases of chronic spondylitis due to other obscure etiology and usually known by such terms as spondylitis, chronic hypertrophic arthritis, etc.; and finally no case of luetic infection.

Such cases of acute osteomyelitis of the spinal vertebræ are comparatively small in number. The paucity of numbers is due in largest measure to the lack of complete knowledge of the manifestations of acute osteomyelitis of the spine among the profession, because of which the diagnosis is not made in many cases. A wider diffusion of knowledge concerning the pathological facts and the clinical possibilities seems necessary; and, undoubtedly, as a consequence of this, correct diagnoses will be made of this condition in those hitherto obscure cases, and reports of cases of this type will increase in number.

There is very little American literature upon the subject. The European literature is considerably more extensive and contains a number of communications published at intervals, in which studies were made of comparatively large series of cases at the larger continental clinics. The remaining papers consist mostly of case reports.

The first case of osteomyelitis of the spine was reported by Lannelogue in 1879. The first important communications were made by O. Hahn in 1895 and in 1899. He combined the cases compiled by Frohner with those from the Bruns clinic. In the 661 cases of osteomyelitis of all the bones of the body thus obtained, fifty-one cases of acute osteomyelitis involved the small and irregular bones and, of these, only one case was found to affect the vertebræ. This gives a proportional figure for Hahn's cases of 1:661 as the relative frequency of acute osteomyelitis of the vertebræ at that time—a truly small

and insignificant proportion. Hahn, however, calls attention to the great probability of many of the cases not having been diagnosed.

Hahn summarizes the result of his investigations as follows:

(1) Acute osteomyelitis of the vertebræ does not form any exception when compared to the other bones, as far as occurrence, etiology and progress of the disease are concerned; however, in many cases it results in severe complications, which are brought about by the extension of the suppuration to neighboring cavities of the body and to the central nervous organs.

(2) It is possible to make a diagnosis in most of the cases if the characteristic symptoms are taken into consideration. The establishment of a diagnosis may be made very difficult if there is an early extension upon the spinal cord and brain, or if there are other complications, or a pyemia, which sets in early.

(3) The prognosis is serious, dependent upon the character of the infection and upon the general condition of the patient, the seat of the disease on the different portions of the spinal column and on the different parts of the vertebræ, and on an early recognition and interference.

(4) The therapy should be as early an interference as the difficulty in establishing a diagnosis in the individual case will permit. It should be active as far as possible. It is limited, however, in those cases in which the process has extended already as far as the central organs, or in which other severe complications exist, especially when pyemia has already set in.

Makins and Abbott's paper in 1896 is a very extensive and excellent review of the subject up to that time and includes all of its phases.

Daverne's thesis, published in 1903, is a very complete review taking in Hahn's work also. He refers to and quotes extensively from the observations of Chipault and of Bergmann and Mickulicz.

Donati's paper in 1906 summarized the experience of the European clinics up to that time. Donati collected fifty-five cases from the literature and added one personal case.

Goebell's paper in 1910–11 paid special attention to the spinal meningeal complications of this disease.

Volkmann's communication in 1915 came next. On the basis of eighty-seven cases from the literature and four personal cases he made an exhaustive study of the disease.

Schwartz's paper in 1920 paid special attention to the spinal cord complications of osteomyelitis of the spine.

All of these men recognized two important factors: (1) the difficulties in making diagnosis in all obscure cases; and (2) the danger of the disease because of its potential complications within the spinal canal. All of the writers recognized the fact that osteomyelitis was part of a general infection (pyemia).

Frequency.—The relative frequency of vertebral osteomyelitis among all forms of acute osteomyelitis in general is rather difficult to determine from the reports in the literature owing to the fact that the cases reported are gathered from a great number of sources, and in none of the reports is there given any basic figures from which the proportion could be calculated. In the last ten years there have been treated at the Mount Sinai Hospital 578 cases of osteomyelitis of all kinds and varieties. Among these there were nine cases of osteomyelitis of the spine—a proportion of 1.5 per cent. The difficulty with these figures, as was pointed out repeatedly by the other men, is that undoubtedly some cases are not diagnosed as osteomyelitis of the

spine, but are classified in the records as cases of general infection (pyemia, bacteriæmia, etc.). At any rate, an important fact to remember is that acute osteomyelitis, while an unusual lesion in the bones of the spine, nevertheless exists and one should be on one's guard for its discovery.

Sex.—In Volkmann's series 72 per cent. of the cases were in men. According to Donati's figures 68 per cent. occurred in men. In our series 55 per cent. of the cases were in men.

Age.—The spread of the disease among the various decades of life is shown in the following table:

Age	Donati Cases	Daverne Cases	Makins and Abbott Cases	Mt. Sinai Cases
Up to 10 years	15	12	5	2
10 to 20 years	24	6	11	
20 to 30 years	11	9	3	3
Over 30 years				4

Fifty per cent. of Volkmann's cases were between eight and seventeen years of age. Hence, in common with all cases of acute osteomyelitis, most cases of osteomyelitis of the spinal vertebræ occurred during the period of adolescence.

Distribution.—The distribution of osteomyelitis of the spine among its various segments is shown in the following table:

Region	Daverne Cases	Makins and Abbott Cases	Mt. Sinai Cases
Cervical	7	3	2
Dorsal	12	5	2
Lumbar	17	10	4
Sacral	5	3	1

Pathogenesis.—Acute hematogenous osteomyelitis is a metastatic lesion during the course of a bacteriæmia, the latter resulting from an acute bacterial lesion on a surface of the body which forms the portal of entry for the infection. In this conception a surface of the body includes not only the skin, but also the entire mucous membrane of the alimentary tract, the genito-urinary tract, etc. The common surface lesions include not only furuncles, carbuncles, etc., on the skin, but also easily demonstrable lesions in the tonsils, and in other lymphadenoid collections lying in the mucous membrane of the pharynx, as well as less demonstrable lesions, such as those in the Peyer's patches.

The fundamental cause of the spreading of the original lesion in the form of metastatic or subsidiary lesions is an infected thrombus lying in the original area of infection, and communicating at some point with the freely circulating blood. Organisms, growing on the surfaces of the thrombus, are discharged,

or pieces of the thrombus itself break off and are discharged into the circulation and, becoming lodged for various reasons in the vascular network of various parts of the body, give rise to secondary lesions. Bone tissue, because of its peculiarities in vascular structure, seems particularly prone to the blocking of these thrombo-emboli and the susceptibility to this is particularly increased during the period of growth when the individual bones contain well-marked divisions into diaphysis and epiphysis.

The various accessory causes, such as trauma, that determine the localization of a secondary focus of infection—fixation point—in a given bone, are associated with accidents in the local circulation which facilitate blocking of any bacterial thrombus-embolus. The essential nature of the pathologic process that develops at the fixation point is a thrombo-arteritis or thrombophlebitis, and the process in the vertebræ is exactly similar to that in other bones in which a dominating position is assumed by the secondary vascular thromboses which must necessarily occur in such a pathological lesion. The all-important secondary effect which these thromboses produce are disturbances of essential nutrition which lead to the death of certain bone cells and the consequent necrosis of certain areas of bone tissue. In the vertebræ these secondary effects occur in the minimum degree because of the characteristics of the local circulation, as will be pointed out subsequently.

The lesions of acute osteomyelitis in the spinal vertebræ are understood only in the light of complete knowledge of the anatomical structure of the bones and of the arrangements of the vascular supply of the individual bones.

The anatomy of the spinal vertebræ is well understood. The heaviest part of the bone is formed by the body which forms the anterior and major portion of the structure. The posterior part is formed by the pedicles on either side which, proceeding backward and toward the median line as the laminæ, serve to form enclosure in which the spinal cord is lodged. The transverse processes and the spinous processes are developmental or rudimentary structures which serve for the most part for the attachment of the various muscles. The heaviest vertebræ are in the lumbar region. That part of the completed spinal column which is most mobile is the cervical portion; next in order come the lumbar vertebræ; in the sacrum the vertebræ have become fused together to form single structure.

Anatomically the vertebræ are formed chiefly of cancellous bone. The body is composed of exceedingly light spongy tissue having a thin coating of compact tissue on its exterior perforated by numerous openings for the ingress and egress of its numerous nutrient vessels. The arch—the pedicles and laminæ—have an exceedingly thick covering of compact tissue and the amount of cancellous bone in the interior is negligible or, in some, entirely absent.

An important item in which the vertebræ differ from most other bones is found in the structure of its periosteum. A true membrane resembling those which are typically found in other bones is wanting. This seems to be necessary because of the mechanical principles upon which the spine, as a whole, and its muscular attachments are built. The superposition of individual bones of comparatively small size to form a rigid and at the same time flexible stem, upon which the rest of the body is hung and supported, makes necessary the complicated joint interlockings which are so typical of the individual vertebræ and of the spine as a whole. Rigidity is obtained by heavy ligamentous structures which hold the bones together and which find and make for the security of the spine as a whole by making deep anchorages in the bone tissue. These ligamentous insertions cover the major part of the entire vertebral surface on the outer and inner side, and the inter-

vening portions are occupied by the tendinous attachments of the spinal muscles which likewise are deeply inserted into the osseous tissue. These tendinous, fibrous and ligamentous structures take on the functions of the periosteum. Their anatomical arrangements are of maximum importance in determining the paths along which suppuration will spread. This will be discussed subsequently.

The blood supply of the bodies of the vertebræ is larger than that for the pedicles and the laminæ. There is a very abundant vascular arrangement for the vertebral bodies arising by a large number of vessels corresponding roughly in number with the number of spinal segments. These are derived from neighboring large main trunks—the basilar in the neck, the intercostal and other branches in the thorax, and the lumbar vessels in the loin. There is a double arrangement—corresponding to the external aspect of the vertebræ and to the interior of the spinal canal. One group of vessels perforate the bodies of the vertebræ from the outer side and break up into a network which supplies the appropriate parts of the bone with blood. These finally anastomose with the network derived from those branches which, having entered the spinal canal, ramify in its interior. Those on the exterior of the bone follow no readily classifiable scheme. The arrangement in the interior of the spinal canal follows the following plan.

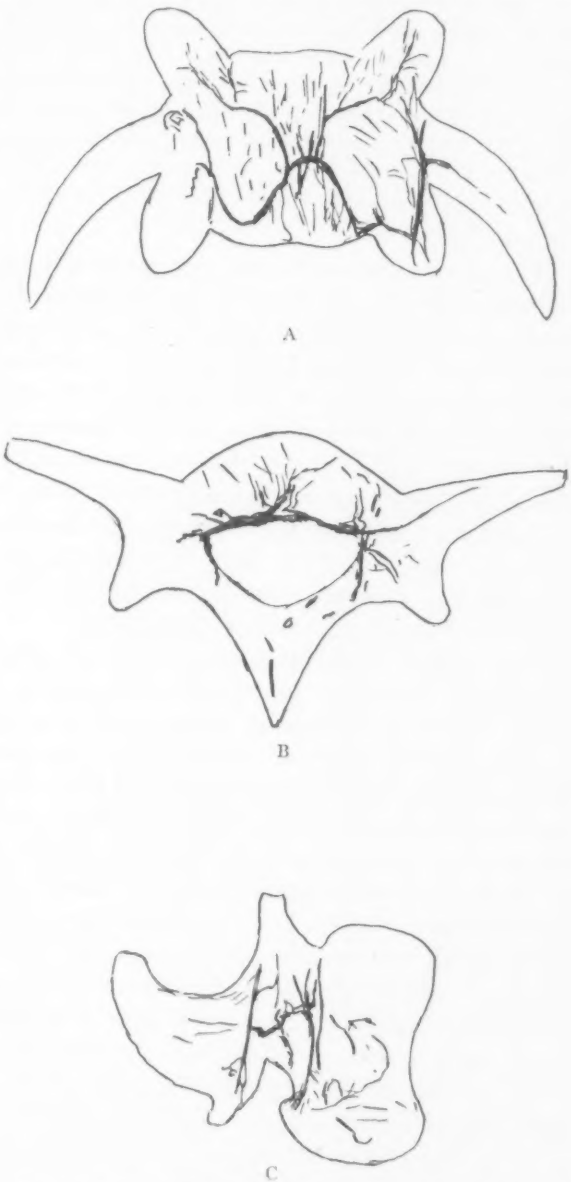

FIG. 1.—Injection specimens showing blood supply of a typical vertebra. A shows the blood supply when viewed anteriorly. B shows the blood supply when viewed superiorly. C shows the blood supply of the anterior of the spinal canal when viewed from the side. Taken from Kuliga and Turck.

The lateral spinal branches enter the spinal canal through the intervertebral foramina and divide into two branches. Of these, one passes along the roots of the nerves to supply the spinal cord and its membranes, anastomosing with the other arteries of the spinal cord; the other divides into an ascending and a descending branch, which unite with similar branches from the artery above and below, so that two lateral anastomotic chains

are formed on the posterior surface of the bodies of the vertebræ near the attachment of the pedicles. From these anastomotic chains branches are given off to supply the "periosteum" and the bodies of the vertebræ, and to communicate with similar branches from the opposite side; from these latter small branches are given off which join similar branches above and below, so that a central anastomotic chain is formed on the posterior surface of the bodies of the vertebræ.

The blood supply of the arches—the pedicles, laminæ and processes—is very much less abundant than that of the bodies and, except for some small perforating branches which are derived from various muscular arterial branches in their immediate neighborhood, the greater part of the blood supply proceeds from the terminal anastomosing ramifications of the network formed from the spinal arteries in the interior of the spinal canal.

Because of these essential anatomical and vascular conditions, thromboembolic lesions in the vertebræ do not follow any particular plan. Lesions are produced which are of irregular size and shape and the size and extent of the latter are determined by the vascular collateral circulation. The involvement is generally of comparatively small size, and more frequently affects the arches and processes of the bones in the lumbo-dorsal region and the bodies of the vertebræ in the cervical region. According to Donati the spinous processes are affected most commonly, the articulating processes next, and the transverse processes least frequently. In three-quarters of the cases in our experience only one vertebræ is affected. According to Bergmann and Mikulicz the disease is not often limited to a single vertebræ; Chipault believes that this occurs only in the sacrum.

The actual involvement is generally of small size. Generally only the "periosteal" layers or the superficial lamellæ of the bone are involved; less commonly the focus extends more deeply into the bone. Owing to the free vascular anastomosis, the formation of sequestra is not constant; and the latter are commonly of inconsequential size. The formation of abscesses is much more common; the mechanism of their formation is that of subperiosteal abscesses in general, as described previously. Depending upon the directional tendency of the infection in the various divisions of the vertebræ, the abscesses have the tendency to extend backward or forward. The paths along which suppuration spreads are well defined because of anatomical conditions and can be classified along the following lines:

A. Suppuration on the exterior surface of the vertebræ.

B. Suppuration in the interior of the spinal canal.

A. In the various segmental regions of the spine, the following pathological-anatomical details can be distinguished:

AA. The Cervical Spine.—1. In the cervical region the commonest location for the focus of infection is in the bodies of the vertebræ. Suppuration forming on the anterior surface of the bodies accumulates at first between the bone and the prevertebral fascia. This is one of the causes of the retropharyngeal abscesses which so commonly occur in children. The direction of spread of the accumulation of pus is downward into the posterior mediastinal space of the thoracic cavity, or upward toward the base of the skull.

2. When the focus of infection develops in the lateral pedicles the pus accumulates on the anterior surface and comes forward in the cellular space in front of the anterior scalene muscles and behind the longus colli muscle; the abscess points in the posterior triangle of the neck being diverted in a lateral direction by the prevertebral fascia.

A similar course is followed by a focus developing on the anterior aspect of the transverse process. More commonly the abscess spreads in the cellular space between the anterior and middle or between the middle and posterior

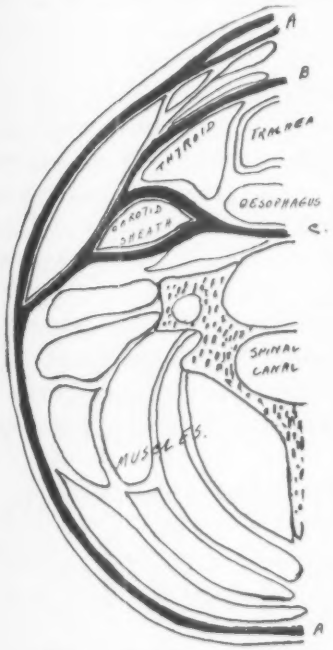

FIG. 2.—Hamisection of the neck; taken from Gray's Anatomy. The heavy lines indicate the main fascial planes. A-A, indicates the deep fascia; B, the pretracheal layer of the deep cervical fascia; C, the prevertebral layer of the deep cervical fascia. Compare with text.

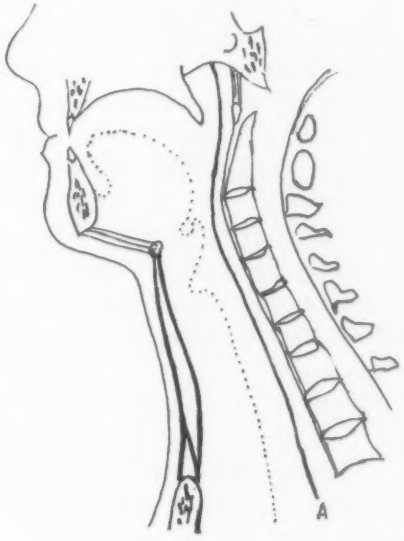

FIG. 3.—Vertical section of the neck and superior mediastinum taken from the *Army and Navy Manual of Surgery*. A is the prevertebral layer of the deep cervical fascia showing the path by which suppuration can spread into the mediastinum.

scalene muscles and points in the posterior triangle of the neck in the interval between the sterno-mastoid and trapezius muscles.

3. Abscesses developing from foci in the posterior aspects of the transverse processes, the laminæ, or the spinous processes, spread backward into the muscle spaces, lodging the various bundles on the posterior aspect of the neck. The commonest one, because of its position, develops in the thickness of the semi-spinalis colli muscle. Deep-seated abscesses are the rule which develop comparatively slowly and these when incised are reached far down in the tissues of the neck.

AB. The Thoracic Spine.—1. In the thoracic spine the commonest location for the foci is in that part of the spine posterior to the bodies. Suppuration developing on the anterior aspects of the pedicles and lateral processes,

in the lower half of the thorax, has the tendency to become enclosed in the fascial sheath of the iliopsoas muscles, or at least to be guided downward and outward along the tendinous origins of these muscles, and to appear clinically as iliopsoas abscesses. Further inward toward the median line the pus accumulates in the posterior mediastinum. Retropleural (extrapleural) abscesses have also been described.

2. Abscesses developing from foci in the posterior aspects of the transverse processes, the laminæ or the spinous processes, naturally spread backward in the depths of the spinal muscles lying in the two grooves between the bend of the ribs and the lateral-posterior aspects of the laminæ and spinous processes. Deep-seated abscesses are the rule.

3. Suppuration derived from foci in the bodies of the vertebræ which develop on their exterior surface accumulate in the cellular spaces of the posterior mediastinum. An acute mediastinitis follows which spreads rapidly upward and downward in this important space; or the process localizes as a mediastinal abscess. Occasionally the mediastinal abscesses discharge into neighboring lymphatic spaces of which the two following are the important ones:

a. A suppurative pleurisy results either from extension of the infection into the pleural sac or because of direct rupture of pus into the latter. It should be remembered that some of these suppurative pleurisies are undoubtedly of hematogenous origin and do not result from extension of the purulent focus in the neighboring spine.

b. A suppurative pericarditis results in ways similar to those attending the formation of a suppurative pleurisy.

4. Combinations of these three forms of pus accumulations have been described. Commonly a suppurative pericarditis is associated also with empyema on one or both sides.

AC. The Lumbar Spine.—1. Foci of osteomyelitis develop most commonly in the transverse processes and arches and in the spinous processes. Suppuration forming on the anterior surface of the transverse processes, or pedicles of the lumbar vertebræ, or on the anterior aspects of their bodies, occasionally localize themselves in these locations and form masses which can be palpated in the depths of the abdominal cavity. The suppurative processes extend forward and to either side in the following ways:

a. High up under the diaphragm on the right side the abscess spreads out under the diaphragm to form a subphrenic abscess. I remember one case in my own experience in which this happened. I have no notes of this case, however, and the details are very indistinct. On the left side the anatomical relations of the left lobe of the liver would make this more likely to become fused in the category of perinephritic abscesses.

b. On either side somewhat lower down the lateral spread of the suppurating process would appear clinically in the form of perinephritic abscesses.

Clinically sufficient spread of the abscess would cause difficulty in making

these fine anatomical distinctions and the probabilities are that all of these would be classified clinically as intra-abdominal abscesses.

2. Suppuration forming in relation to the posterior aspects of the arches and the transverse and spinous processes develop posteriorly in the depths of the spinal muscles in the small of the back.

Clinically and before these abscesses are opened they can be confused with those abscesses developing in the perinephritic space described in the preceding paragraphs which have come to the surface in the costo-vertebral angle. The differentiation should, however, be immediately made when the abscesses are opened.

3. Cases of peritonitis have been described complicating osteomyelitis of the lumbar vertebræ. The paths of infection include (*a*) extension by the lymphatic vessels into the peritoneal cavity; (*b*) frank rupture of an abscess into the peritoneal cavity; and (*c*) hematogenous (metastatic) infection of the peritoneal space.

AD. The Sacrum and Coccyx.—1. Suppuration derived from foci of osteomyelitis developing on the posterior aspect of the sacrum and coccyx appears clinically as localized abscesses over the sacrum or lower part of the spine and are practically subcutaneous affairs. These are very simple matters.

2. Suppuration developing from foci of osteomyelitis on the anterior aspect of the bones of the sacrum and coccyx develops in the hollow of the sacrum and spreads forward, laterally and downward. The following paths can be distinguished anatomically:

a. Suppuration developing from the lower segment of the sacrum or from the coccyx gathers in the interval between the coccyx and anus and lies beneath the levator ani muscle and the anal fascia. The pus spreads out of the pelvic cavity through the sacrosciatic notch and turns up in the depths of the gluteal muscles as gluteal abscesses. Or the pus burrows downward and appears clinically as a para-anal abscess.

b. Another variety develops above the line of attachment of the levator ani muscle. In the larger number of these a subperiosteal abscess develops on the inner aspect of the bone and between it and the iliacus muscle, and spreads upward to the crest of the ileum, where it appears as an extra-peritoneal abscess; some of these point above Poupart's ligament. In a lesser number abscesses form which spread forward and downward in the hollow of the sacrum and between the bone and the obturator and recto-vesical layers of the pelvic fascia. The pus gathers in the ischiorectal fossa and appears clinically as an ischiorectal abscess. A certain number of these spread upward, so that they cannot be palpated from below through the rectal canal, or at least are palpable with great difficulty. These are the cases which most frequently appear clinically as extremely obscure forms of general infection (pyemia, sepsis, etc.).

Suppuration in the Interior of the Spinal Canal.—Inflammatory changes in the interior of the spinal canal may be limited to an outpouring of inflammatory exudate and to a thickening of tissues lying between the dura mater

and the bone—a pachymeningitis externa. Suppuration, however, occurs very commonly and takes the form of an accumulation between the bone and spinal dura mater. This corresponds pathologically to a subperiosteal abscess. The accumulation may remain localized or may rupture through the dura and into the subarachnoid space; a spinal meningitis results. In this sense the latter is entirely independent of any form of meningitis which results from a hematogenous infection of the meninges during the course of any bacteriæmia which may be present. Cases have been described in which the extradural abscess has ruptured and escaped through one of the spinal foramina leading to the formation of a simple or complex abscess on the exterior of the spine in and between the muscle planes and bundles.

Compression of the cord leading eventually to a paraplegia may be entirely due to any of the causes suggested, *i.e.*, pachymeningitis externa or extradural abscess. Transverse and ascending forms of myelitis are described.

(*To be continued*)

OSTEOPOROSIS OF THE HUMERUS FOLLOWING FRACTURE

By Nicholas S. Ransohoff, M.D.
of New York, N. Y.
From the Hospital for Joint Diseases

February 28, 1926, Mrs. B. E., age forty-one, presented herself at my office after having had a fracture seventeen days previously, in the middle third of her left humerus. While skiing at Lake Placid, the patient fell on her outstretched arm and heard a snap. Amidst a panicky crowd no first aid was administered, and the arm was allowed to swing in the winds unprotected for about two hours, until she arrived at a hospital.

Here an X-ray photograph was taken (Plate 1). An anæsthetic was given and the fracture was reduced. A practically perfect reduction was obtained and the fragments held in opposition by means of circular splints, in combination with a pyramidal cushion in the axilla.

She recovered quite rapidly from the shock of the accident and came to New York, at which time I first saw her. The original splints were left intact for another few days, then, because she had rather more pain than would ordinarily be expected, diathermia was given through the fracture site. The electrodes were applied anteriorly and posteriorly. This gave so much relief that it was continued for several weeks. When removing the splints for this purpose, it was noticed that the arm had about the usual amount of swelling and a moderate amount of ecchyomosis, more marked on the inner side. There was definite tenderness over the point of fracture which was just about in the middle third of the humerus. Otherwise no abnormality was noticed.

At the end of several weeks, March 16, 1926, because palpation gave no evidence of union, a second X-ray picture (Plate 2) was taken to determine the amount of callus production. The X-ray report as given by Dr. Charles Gottlieb was: "Radiographic examination of Mrs. B. E. shows an oblique fracture through the left humerus, somewhat above its midpoint. . . . There is no evidence of callus formation or union." However, after the next set of films was taken it was noticed upon closer study of this first one that some structural change was taking place in the distal fragment. This showed itself as a peculiar mottling of the bone more marked distally, which, for want of a better term, we shall call "osteoporosis". The corticalis was well marked and had a very slightly honey-combed or moth-eaten appearance. There seemed to be no periosteal reaction whatever. The proximal fragment showed somewhat the same appearance at its distal end.

April 6, 1926, a third set of radiographs were taken (Plate 3). These showed what Doctor Gottlieb reported as "spotted atrophy". There was a very startling increase of those processes, which must have been in their incipiency in Plate 2. The bone looked like a cross section of sponge rubber. The honey-combing was markedly increased. The corticalis had lost a good deal of its definition. The areas which were most permeable to the X-rays were much more numerous and more definitely defined. There was a small amount of callus production. The rarefaction at the distal end of the proximal fragment was not as marked as that in the distal fragment, and the proximal end remained normal.

At the suggestion of Dr. Leo Mayer, who saw the patient in consultation at this time, the skull, right shoulder and humerus and left radius and ulna were then radiographed (Plate 5), and all the radiographs were reported negative. The X-ray plates of April 28 (Plates 4 and 5), three weeks later, show a still greater increase of the rarefying process throughout the distal fragment. Despite this increased atrophy, the site of

PLATE 3.—April 6, 1926.

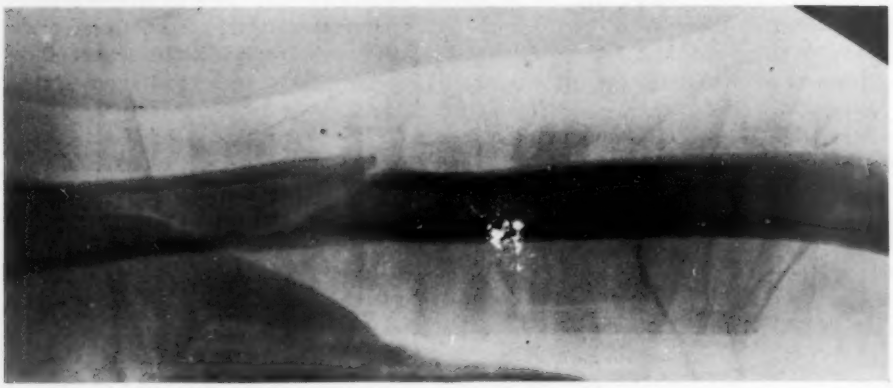

PLATE 2.—March 16, 1926.

PLATE 1.—Taken two hours after injury. Showing unreduced fracture. February 11, 1926.

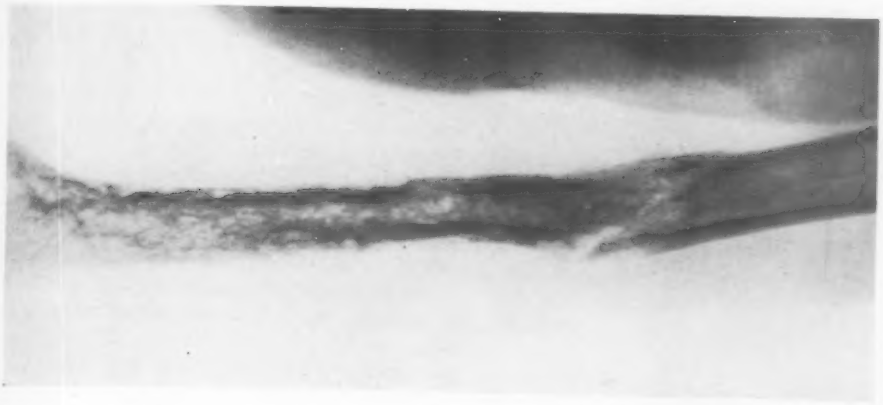

PLATE 4.—April 28, 1926.

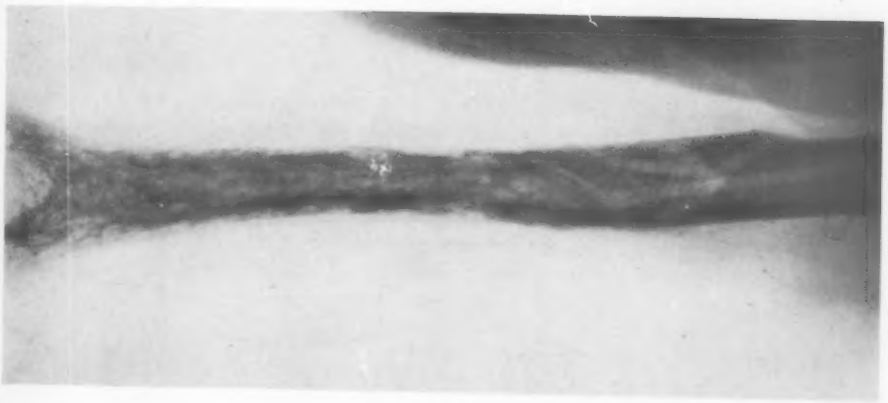

PLATE 5.—April 28, 1926.

PLATE 6.—April 9, 1926. Note normal radius and ulna.

PLATE 1.—Taken two hours after injury. Showing unreduced fracture. February 11, 1926.

PLATE 2.—March 16, 1926.

PLATE 3.—April 6, 1926.

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PLATE 7.—July 26, 1927.

the fracture was then enveloped in a well-developed callus. The atrophy in the distal extremity of the proximal fragment was well diminished.

The final plate (Plate 7), taken July 26, 1927, seventeen months after injury, shows a normally healed fracture of the humerus and complete disappearance of the pathological changes noted in the previous pictures.

After the third set of radiographs, inasmuch as we had never seen a condition which looked like this, we began to grope for the etiological factor. The Wassermann reaction was negative, with both alcoholic and cholestrinized antigens. The basal metabolism was within normal limits. The Goetsch test was negative. Blood chemistry as done by Doctor Bernhard, gave the following figures:

Urea Nitrogen	14.7
Creatinine	0.5
Sugar	0.80
Calcium (Mil per 100 c.c. of serum)	0.2
Magnesium (Mil per 100 c.c. of serum)	2.4

All these figures were within normal limits. At the time that the patient was seen by Dr. Leo Mayer he suggested that the whole process might be explained on circulatory grounds, because the fracture was at approximately the point which Lexer has demonstrated as the entrance point of the nutrient artery of the humerus (Plate 8).

When further investigation of the blood supply of the humerus was made, it was found that Gray describes the nutrient artery as entering the bone on "the medial margin just below the attachment of the coraco-brachialis". The foramen points downward. Braus in his anatomy has a simple diagram which illustrates the course and development of the nutrient artery very well (Plate 9).

Upon investigation of the experimental work which has been done by so many on the healing of fractures, and the regeneration and the degeneration of bone, no really adequate explanation of what had occurred can be found, except that of Johnson. He shows by his experimental work that the diaphysis of the long bones is dependent for its nutrition upon three sets of vessels. These are in order of their importance: (1) Nutrient artery; (2) Metaphyseal vessels; (3) Periosteal vessels.

Johnson concludes: 1. "Nutrient vessels maintain viability throughout the medulla and supply the inner half of the cortex. Repair is active when the nutrient vessels are intact."

2. "Metaphyseal vessels maintain viability

PLATE 8.—The arteries of the humerus of a new-born child injected with mercury. (X-ray picture photographed from Lexer, Kuliga and Turk.)

throughout the medulla and inner half of the cortex, but repair is not as active as in the controls except close to the metaphyseal ends, being noticeably delayed in the middle region of the shaft."

3. "The periosteal system does not normally supply more than the outer half of the cortex, and is unable to afford effectual collateral supply to the medulla of the diaphysis under four weeks' time. The periosteal repair is relatively poor in the healing of cortical defects."

Based upon these observations a theory has been evolved as the explanation of the process which has taken place in this case: *i.e.*, the site of fracture was in all probability at the nutrient foramen and therefore because there was no immobilization for fully two hours subsequent to the fracture, the nutrient artery must have been severely lacerated. Furthermore, because of this same trauma, the periosteum was badly lacerated. The metaphyseal vessels which Johnson says play so great a part in bone repair in young dogs are, in the adult, probably non-existent, or so small as to be unable to form an adequate anastomosis. It therefore seems fair to assume, that there has been very marked devitalization of the distal fragment due to interference with the blood supply. This probably has not been so great as to cause actual death of the bone, because on examining the X-ray plates of bone grafts, these grafts which surely die, do not present the picture we see in this case.

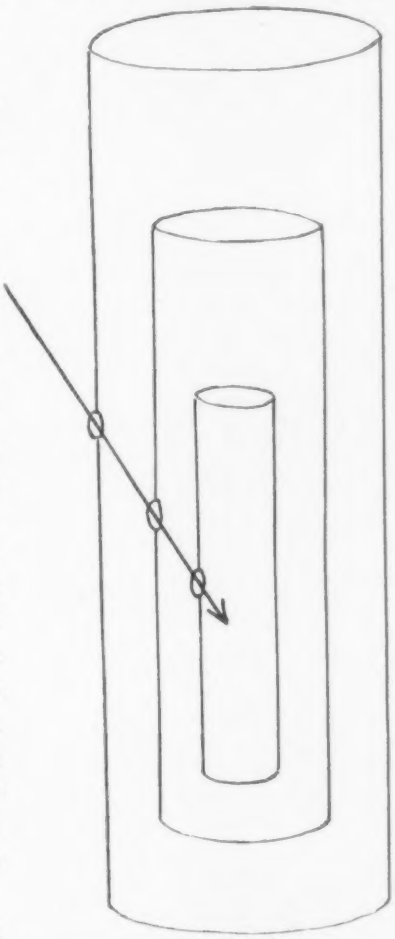

PLATE 9.—Direction of the Nutrient Canal of the Humerus. Three sizes of the growing humerus (placed one on top of the other). The relative positions of the nutrient artery is shown in this way. The proximal end grows faster than the distal. The nutrient foramen remains in the middle of the three different sizes. (Redrawn from Braus.)

Whether or not there was some trophic nerve disturbance along with the circulatory disturbance, it is exceedingly difficult to say, but in view of the fact that there was no other evidence of any injury, it does not seem fair to assume this. The repair was probably effected through an anastomosis between the periosteal vessels and the undamaged part of the nutrient artery which supplied the upper fragment.

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ON THE TREATMENT OF THE SEPARATION OF THE LOWER EPIPHYSIS OF THE FEMUR *

By Ernst Gold, M.D.
Of Vienna, Austria

The most frequent separation of an epiphysis is at the lower end of the radius. In the material seen at the accident department of the Eiselsberg Clinic, in Vienna, 25.9 per cent. of the fractures of the lower end of the radius up to the eighteenth year were epiphyseal separations. On the contrary epiphyseal separation of the lower end of the humerus is a rare condition, while fractures of the humerus above the elbow are frequent in childhood. The most important epiphyseal separation of the lower extremity is that of the lower end of the femur. There are statistics (Brun, Kirmisson) which indicate them to be most frequent of all epiphyseal separations (28 per cent.).

Fig. 1.—Traumatic epiphyseal separation at lower end of femur, before reduction. Lateral view.

The complete separation of the lower epiphysis of the femur is the most important from the point of view of the treatment, because it is often followed by premature ossification of the epiphyseal line with cessation of the growth of the femur, especially when the accident happens to take place in early childhood. On the other hand, in a case of an incomplete dislocation of the lower epiphysis of the femur of a young boy of fourteen years we found, although the X-ray picture shows marked dislocation, an absolute functional recovery. One year after injury the boy is the fastest runner in his class at school. Complete

* Presentation made at a clinic held September 1, 1928, for the Travel Study Club of American Physicians.

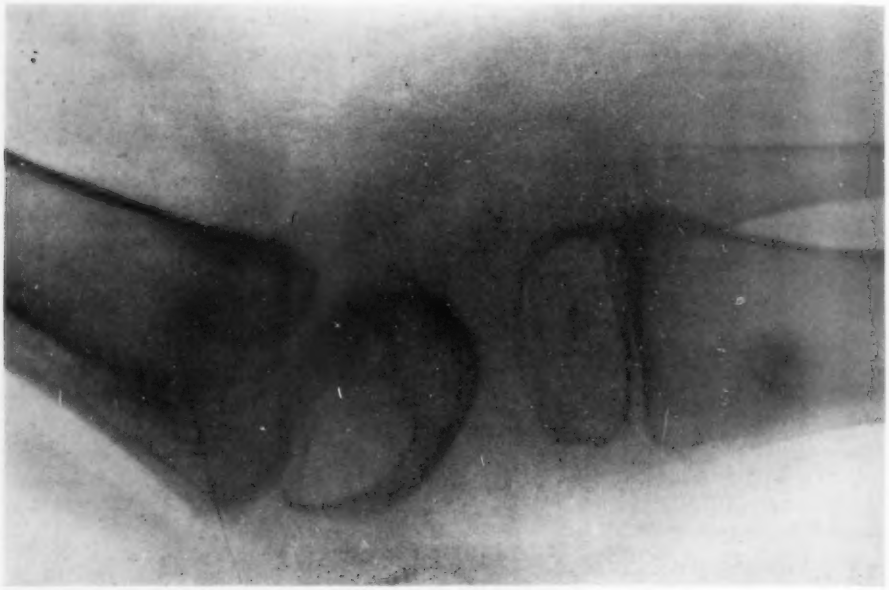

Fig. 3.—Epiphyseal separation at lower end of femur, after reduction. Lateral view.

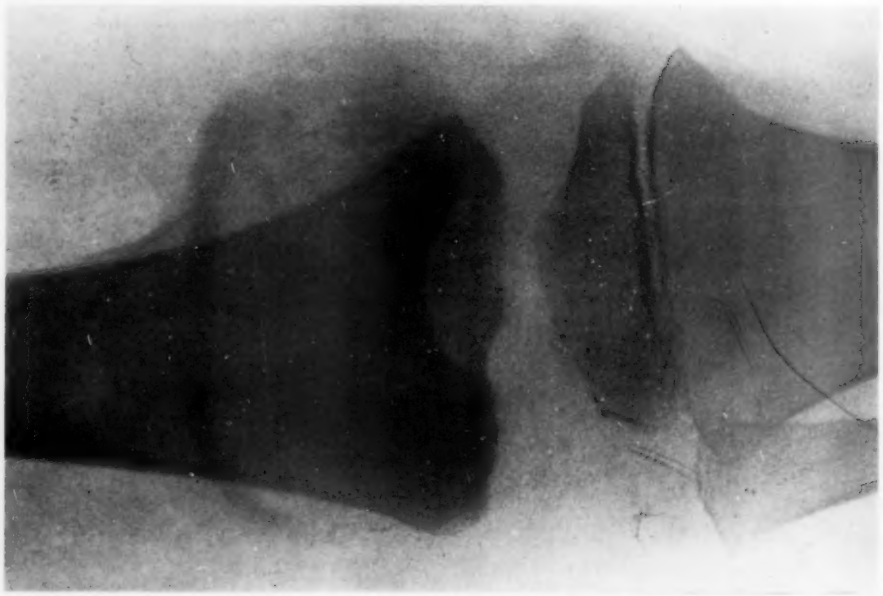

Fig. 2.—Epiphyseal separation at lower end of femur. Antero-posterior view.

dislocation of the lower epiphysis of the femur happens most frequently from a forcible twist of the leg, as when caught in a turning wheel. Epiphyseal separation is always associated with traumatic elevation and partial rupture of the periosteum.

Figures 1 and 2 are X-ray pictures of the knee of a boy of six years whose leg was thus caught by a wagon-wheel and forcibly twisted. Immediately after the injury he was taken into a country hospital where the case was diagnosed as ligamentous strain with bleeding within the joint, and it was treated with ice-bags. The practitioner felt sure that the injury of the knee-joint was severe, although the X-ray findings were apparently negative, and he suggested the possibility of epiphyseal separation. At the examination in the accident department four weeks after the original injury the injured leg was found to be three centimetres shorter than the other one with slight varus position, recurvation and marked lateral mobility of the knee. In the popliteal space a big bony prominence was felt. In the X-ray pictures, which were done in the station, there was a complete absence of the lower epiphysis of the femur from its normal position in the antero-posterior view. The lateral view showed the whole lower epiphysis of the femur to be separated from the diaphysis and dislocated forward into a position behind the patella, with the articulating surface turned toward the posterior surface of the patella and still attached by periosteum and new formed callus to the shaft. The boy was not able to bear any weight on the leg. At first I attempted to make a closed reduction without success. Then an open operation was resorted to. A longitudinal incision was made in the popliteal region along the course of the nerves and vessels which were isolated and retracted. The lower end of the diaphysis was freed from periosteum. The callus, uniting the epiphysis with the shaft, was separated subperiosteally. By this method I was able to avoid opening the knee-joint. In the bottom of the wound the separated epiphysis could be felt. An attempt was now made to reduce the epiphysis by means of direct pressure on the femur from behind forward while strong traction was made on the leg in flexed position. Only a partial reduction of the epiphysis was possible and this could not be maintained. It was decided therefore to attempt a gradual reduction by skeletal traction. A Steinmann-pin was driven through the head of the tibia and the leg was placed on Braun's frame (model of the Eiselsberg Clinic, which is adjustable to length as well as to amount of flexion) in semiflexion. The extension was then made in the line of the axis of the shaft of the femur. An X-ray picture showed the epiphysis gradually moving toward the position of reduction. Further reduction, however, could apparently not be executed without bringing the traction into the axis of the leg, the flexion of the knee being increased at the same time. A series of X-ray pictures on consecutive days showed a gradual movement of the epiphysis to complete reduction. Examination one year after reduction showed the boy to have regained a complete function with exception of a slight limitation of extension of the knee. He shows no symptoms of premature ossification of the epiphyseal line. The same method was applied in a fresh case of epiphyseal separation of the femur with perfect recovery. It is also applicable in the treatment of fractures above the elbow and has been used in several cases with complete success. This case of epiphyseal separation seems to be worthy to be reported because of the fact that a principle of treatment was used in the reduction which is not often seen.

FIG. 2.—Epiphyseal separation at lower end of femur. Antero-posterior view.

FIG. 3.—Epiphyseal separation at lower end of femur, after reduction. Lateral view.

FRACTURES OF THE TIBIA INVOLVING THE KNEE*

By Herbert S. Steuer, M.D.

of Cleveland, Ohio

From the Anatomical Laboratory of the Western Reserve University

FREQUENCY AND CAUSE

In this era of the automobile the human knee is in a position of peculiar disadvantage, for it lies at the level of the bumper. Hence, fractures of the tibia near or involving the knee-joint are becoming more frequent and, in view of the consequent disability, more important.

Current text-book accounts, derived from the accidents of an earlier day (see literature cited), dwell upon indirect violence as the more common method of producing a fracture of the tibia involving the knee-joint. In the few articles recently published upon the subject (see literature cited) direct violence is accorded the premier rôle. Both views are correct for the particular date of their expression, but we must expect fractures from direct violence to claim our attention ever more insistently.

During the past fifteen years, among 1300 bodies of our deceased fellow-townsmen delivered to the Medical School and now housed as skeletons in the Hamann Museum are sixty-six fractured tibiæ. Thus fracture of the tibia occurs in 5 per cent. of a population by no means confined to paupers and vagrants. Our selection is a fair cross section of the Cleveland people, excepting the financially stable.

Almost half the tibiæ, thirty-two in fact, show fracture in the lower third. This is to be expected. Of the rest, eighteen involve the shaft, but do not enter the knee-joint. Thirteen of these fractures shattered the bone in its middle third, five in its upper. The remaining sixteen occur primarily in the upper articular surface itself. It is with this group that we are immediately concerned.

The examples at our disposal clearly arrange themselves into two series. The first, consisting of twelve specimens, involves either condyle or both, varies in extent of injury and in disorganization of the underlying tuberosity, but always results in deformity. It is rarely accompanied by synchronous fracture of any other bone. The second, comprising four examples, shows no such extensive injury, results in no recognizable deformity, and may be secondary to a more severe injury elsewhere in the body.

By the severity, localization and restriction of injury to the knee alone

* This article was completed two days before Doctor Steuer's tragic death in line of duty on June 6, 1928. It has been for me an act of homage to the memory of my colleague to guide the article through the press. By it we may the better realize how promising a recruit has been lost to Surgery.

T. Wingate Todd.

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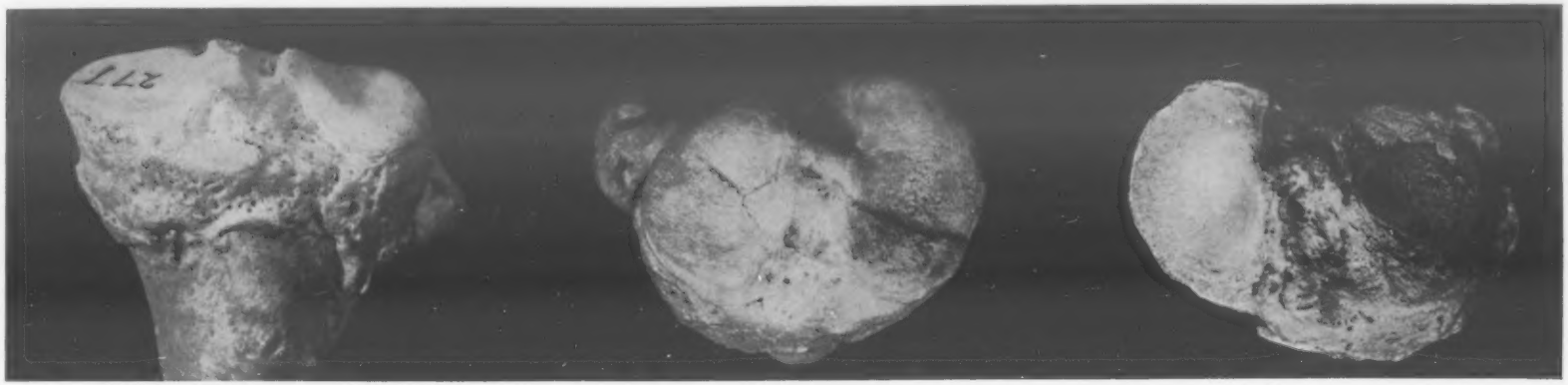

FIG. 1 FIG. 3 FIG. 5

FIG. 2 FIG. 4 FIG. 6

PLATE I.—Group I. Class A. Staves of Upper Extremity.

FIG. 1.—Ventral view. Left Tibia, Fibula. No. 277, Male, White, sixty-seven years. Stave crushes entire upper extremity downward on shaft. FIG. 2.—Anteroposterior Radiogram. Stave seen on medial border. FIG. 3.—Upper Extremity. Right Tibia, Fibula. No. 546, Male, White, about fifty years. Stave similar to No. 277, but with crack running across entire articular face. FIG. 4.—Anteroposterior Radiogram. Deformity of lateral tuberosity and head of fibula. FIG. 5.—Upper Extremity. Left Tibia. No. 1416, Male, Negro, about forty-eight years. Stave below tubercle. Lateral tuberosity partially split off, driven downward and tilted. FIG. 6.—Anteroposterior Radiogram. Deformity of upper shaft and of lateral tuberosity.

it is apparent that the first group is the result of direct violence. In eight of these instances the left bone is injured. Upon similar analysis the injury of the second group is due either to a minor direct violence or to indirect violence, the chief injury being found in vertebral column, pelvis, upper

FIG. 7

688

FIG. 9

1534

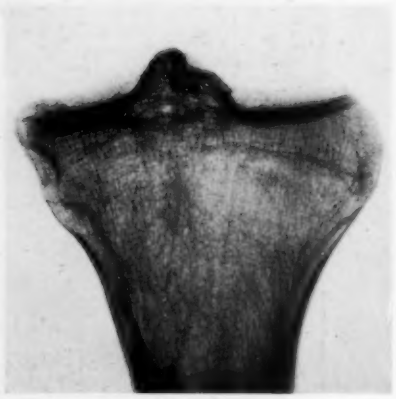

FIG. 8

FIG. 10

PLATE II.—Group I. Class B. Comminuted Fractures with Splitting Off and Impaction of Tuberosity.

FIG. 7.—Upper Extremity. Left Tibia. No. 688, Male, White, twenty-four years. Multiple cracking without real split of either tuberosity. FIG. 8.—Anteroposterior Radiogram. Clinically this would be negative. FIG. 9.—Upper Extremity. Left Tibia. No. 1534, Female, Negro, about forty-five years. Comminuted fracture with splitting off of medial tuberosity. Smaller fragments lost in maceration. Immediate death. FIG. 10.—Anteroposterior Radiogram. Taken soon after death. Shattering of upper extremity of tibia. This is a radiogram on admission, not of the macerated bone.

femur, or foot. A fracture of the calcaneus in one of our specimens occurs in the contralateral bone.

It is instructive to note that Crillovich, who described eleven cases of fracture of the lateral tibial condyle, cites eight as caused by direct violence. Clearly enough then, both from our own experience and from recently published work, direct violence is increasingly responsible for fracture of the upper end of the tibia.

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FIG. 11
1322

FIG. 13
1382

FIG. 15
577

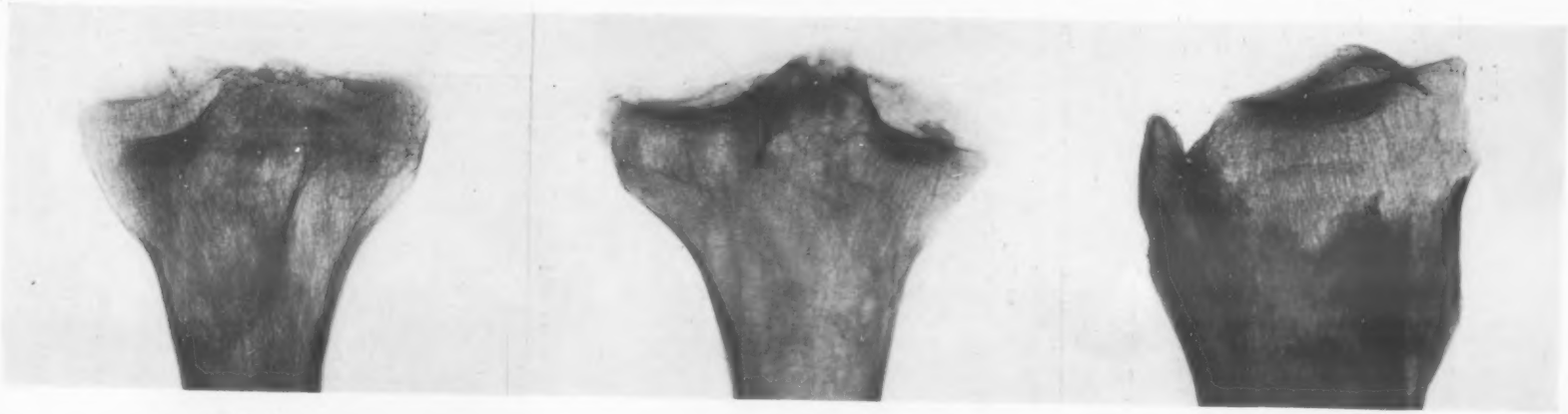

FIG. 12

FIG. 14

FIG. 16

PLATE III.—Group I. Class B. (*Contd.*)

FIG. 11.—Upper Extremity. Left Tibia. No. 1322, Male, White, fifty-four years. Comminuted fracture of both condyles with splitting off and tilting of medial tuberosity. FIG. 12.—Anteroposterior Radiogram. Evidence of deformity, especially of medial tuberosity. FIG. 13.—Upper Extremity. Right Tibia. No. 1382, Male, White, sixty-three years. Comminuted fracture of both articular faces with tilt and impaction of medial tuberosity. Arthritic deposits. FIG. 14.—Anteroposterior Radiogram. Comminution of both tuberosities with tilting and impaction of medial one. Arthritic deposits. FIG. 15.—Ventral Aspect. Right Tibia. No. 577, Male, White, about fifty years. Comminuted fracture of lateral tuberosity complicated by stave of entire upper extremity. Tilting of lateral tuberosity. FIG. 16.—Lateral Radiogram. Note stave. Disorganization of lateral tuberosity not seen.

Physical Features.—It is quite important to describe rather carefully the physical features of these fractures for they are often seen late, long after the fracture is healed. Indeed there may be no definite history of fracture, but only of considerable injury to the knee with swelling lasting for

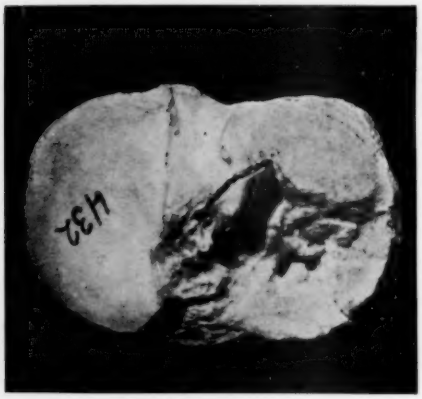

FIG. 17

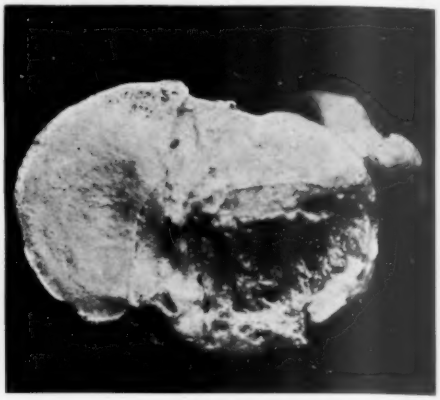

FIG. 19

FIG. 18

FIG. 20

PLATE IV.—Group I. Class C. Depressed Fracture with Disorganization of Substance.

FIG. 17.—Upper Extremity. Left Tibia. No. 432, Male, White, fifty years. Depressed fracture of lateral condyle with disorganization of tuberosity. Note small impacted fragments. FIG. 18.—Anteroposterior Radiogram. Indication of disorganization of lateral tuberosity. FIG. 19.—Upper Extremity. Left Tibia, Fibula. No. 1307, Male, White, eighty-two years. Depressed fracture of lateral condyle and disorganization of tuberosity. FIG. 20.—Anteroposterior Radiogram. Evidence of disorganization of lateral tuberosity.

weeks and permanent or increasing disability. There may even have been a röntgenogram equivocal or negative in its indications. We have seen such cases, coming under observation late, diagnosed and even operated upon for osteitis fibrosa, bone cyst, or tuberculosis. It is the need for more efficient differential diagnosis which prompts the attention given in this article toward the elucidation of an injury increasing in its frequency and importance.

For anatomical reasons the first group further separates itself into three phases of progressive severity. In the first of these (A) there is a stave of

the upper extremity of the tibia, cracking the articular surface more or less. We have three such specimens. In the second (B) there is a comminuted impacted fracture of the tuberosities with a Colles-like deformity. There are five of this class. The third class (C), with four specimens, is char-

FIG. 21
1503

FIG. 23
1591

FIG. 22

FIG. 24

PLATE V.

FIG. 21.—Upper Extremity. Right Tibia. No. 1503, Male, White, about sixty-five years. Depressed fracture and disorganization of lateral tuberosity. Some staving of upper extremity. FIG. 22.—Anteroposterior Radiogram. Marked disorganization of lateral tuberosity. FIG. 23.—Upper Extremity. Left Tibia. No. 1591, Male, White, sixty-two years. Depressed fracture and disorganization of both tuberosities. Partial splitting off of lateral tuberosity. FIG. 24.—Anteroposterior Radiogram. Marked disorganization of both tuberosities.

acterized by a depressed fracture of the condyle and consequent disorganization of the substance of the tuberosity.

GROUP I.—*Direct violence.* CLASS A.—*Staves of upper extremity with splitting and impaction of lateral tuberosity and some involvement of knee-joint.* Three specimens.

No. 277. Male, white, sixty-nine years of age. Left tibia. Impacted crushing fracture canting the lateral condyle downward and laterally. The incomplete fracture through the intercondyloid area is well healed. No deposits of bone. The slight deformity appears on the radiogram.

No. 546. Male, white, about fifty years of age. Right tibia. Crushing impacted fracture similar to No. 277, but cracking both condyles and involving head of fibula. No consequent bone deposits. Deformity shown on radiogram.

No. 1416. Male, negro, about forty-eight years of age. Left tibia. Oblique fracture splitting off and impacting the lateral tuberosity so that the condyle faces backward and

FIG. 25
359

FIG. 27
1506

FIG. 26
821

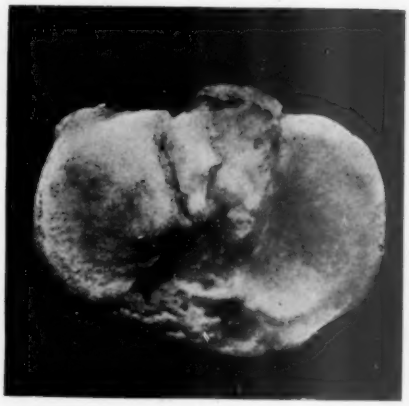

FIG. 28
1284

PLATE VI.—Group II. Minor Fractures.

FIG. 25.—Upper Extremity. Right Tibia. No. 359, Male, White, sixty-three years. Slight transverse crack of lateral condyle. FIG. 26.—Upper Extremity. Left Tibia. No. 821, Male, White, seventy-nine years. Semicircular crack of lateral condyle. FIG. 27.—Upper Extremity. Right Tibia. No. 1506, Male, White, fifty years. Localized comminuted fracture of dorsal part of lateral condyle. FIG. 28.—Upper Extremity. Right Tibia. No. 1284, Male, White, about fifty years. Avulsion of eminentia intercondyloidea.

outward and is displaced downward. It is also deformed by bone deposits. There is an impacted fracture through the upper fourth of the shaft. Deformity admirably shown on radiogram.

In these three specimens the essential fracture is a crushing one staving the upper extremity into the shaft, cracking the condyle and tilting it into a position comparable with that of the radius in Colles's fracture. This class merges into Class B.

CLASS B.—*Comminuted fractures of tibial condyles.* Five specimens.

No. 688. Male, white, twenty-four years of age. Left tibia. Comminuted fracture of both condyles with downward driving and impaction, but no tilting of lateral tuberosity. Very similar to Class A, but without stave of entire upper extremity. No deposits of bone on articular face. Deformity indicated, but not well seen on radiogram.

No. 1534. Female, negro, about forty-five years of age. Left tibia. Comminution of ventral part of both condyles. Complicated by fatal internal injuries. Smaller fragments lost in maceration. Medial tuberosity split off, but not impacted. Condition well shown in radiogram of knee on admission.

No. 1322. Male, white, fifty-four years of age. Left tibia. Comminuted fracture both condyles. Medial tuberosity split off and impacted with Colles-like tilt of dorsal portion which is driven downward and faces inward and backward. No bone deposits on articular face. Deformity shown in radiogram.

No. 1382. Male, white, sixty-three years of age. Right tibia. Comminuted fracture of both condyles with Colles-like tilt and impaction of medial tuberosity which is driven downward and rotated to face somewhat medially. Some bone deposits on articular face which is also crippled by arthritic deposits. Lateral condyle of femur also split and impacted. Deformity shown in radiogram.

No. 577. Male, white, about fifty years of age. Right tibia. Comminuted fracture of lateral condyle only, complicating a stave of upper extremity including tubercle. Impaction of lateral tuberosity which is rotated to face outwards. This specimen shows characteristics of all three classes, but perhaps belongs most obviously to Class B. Deformity well shown in radiogram.

While No. 577 shows a stave like those of Class A the essential feature of it and of all the other four is a comminution of the articular surface, without consequent disorganization of the internal structure of the lateral tuberosity except in No. 577.

CLASS C.—*Fractures of articular face with disorganized structure of lateral tuberosity.* Four specimens.

No. 432. Male, white, fifty years of age. Left tibia. Comminuted fracture of articular face of lateral tuberosity alone with depression of fragments into substance of tuberosity. No splitting off or impaction of tuberosity or stave of upper extremity. Disorganized structure of lateral tuberosity shown faintly in radiogram.

No. 1307. Male, white, eighty-two years of age. Left tibia. Comminuted fracture of lateral articular surface with depression of fragments into substance of lateral tuberosity. No splitting off or impaction of tuberosity or stave of upper extremity. Disorganization of substance of lateral tuberosity shown in radiogram.

No. 1503. Male, white, about sixty-five years of age. Right tibia. Comminuted fracture of lateral articular face with depression of fragments into substance of tuberosity. No splitting off or impaction of lateral tuberosity or stave of upper extremity. Disorganization of substance shown in radiogram.

No. 1591. Male, white, sixty-two years of age. Left tibia. Comminuted fracture of entire articular surface with depression of fragments in substance of tuberosities. Complicated by split of lateral tuberosity, but without impaction of tuberosity or stave of upper extremity. Disorganization of substance shown on radiogram.

In these specimens the essential feature is a crushing of the fragments of the articular face into the substance of the tuberosity. These are the specimens which, when seen late, give rise to the diagnosis of osteitis fibrosa, bone cyst, or tuberculosis. The history of injury combined with the characteristic localized disorganization of substance should, however, clear the diagnosis.

There is not any hard and fast distinction between the three classes. Nor can there be postulated a corresponding difference in the violence of injury. The distinctions are accidental, but the cause is direct violence on the knee itself. Sometimes the force is expended on tibia alone, but it may also affect the femur.

GROUP II.—*Minor injuries.* Four specimens.

No. 359. Male, white, sixty-three years of age. Right tibia. Slight crack in lateral condyle. Similar slight crack in lateral condylar face of right femur. Radiogram negative.

No. 821. Male, white, seventy years of age. Left tibia. Slight crack in lateral condyle. Crushing fracture of right calcaneus. Radiogram of tibia negative.

No. 1506. Male, white, fifty years of age. Right tibia. Comminuted cracking dorsal part of lateral condyle. Medial condyle of right femur also cracked. Extensive fractures of pelvis. Dislocation of right hip. Fatal injuries. Radiogram of tibia negative.

No. 1284. Male, white, about fifty years of age. Right tibia. Cracking and separation of area of attachment of cruciate ligaments. No other fractures. Radiogram of tibia negative except for evidence of bone deposit.

These four specimens are evidently minor injuries of the knee-joint comparable with cracking of the articular surface of the patella found in quite a number of our skeletons. One was an injury limited to the knee-joint. Three were the result of indirect violence mainly expending itself in other areas. Clinically an exact diagnosis, immediate or late, would be impossible, but temporary swelling and disability nevertheless occur.

Distribution.—In Table I are given the numbers of skeletons examined for this type of fracture, arranged by sex and stock. The distribution is, however, erratic, for the table shows white males to be much more frequently injured than females or negroes. This is probably an accident of the series, but it is rather impressive to find that 1.2 per cent. of all our skeletons show fracture of the upper tibial extremity.

TABLE I.

	Total No.	Fractures	Percentage
White males	854	14	1.6
White females	109	0	0.0
Negro males	340	1	0.3
Negro females	97	1	1.0
Total	1310	16	1.2

Table II details the location of the fracture. Our figures, allowing for the errors of random samples, follow closely those of Crillovich. Some 50 per cent. involve the lateral tuberosity alone and in 25 per cent. the entire upper extremity is injured.

TABLE II.

Fracture	Steuer	Per cent.	Crillovich	Per cent.
Both tuberosities	4	25.0	6	25.0
Lateral tuberosity	9	56.5	11	46.0
Medial tuberosity	2	12.5	4	16.5
Eminentia intercondyloidea	1	6.0	3	12.5
Total	16	100.0	24	100.0

Table III is, perhaps, the most striking of all. From this one may infer that, under forty years, agility of the individual usually saves him from injury. The majority of such fractures occur between forty and sixty years. After sixty years the percentage diminishes. This can only mean that fracture in old age, when it occurs, is complicated by other injuries causing death, so that recent fractures are less likely to find their way into our collection. Among our sixteen examples only two died immediately.

TABLE III.

Age	No. of fractures	Per cent. of total
– 9	0	0
10–19	0	0
20–29	1	6
30–39	0	0
40–49	2	12
50–59	6	37
60–69	5	31
70–79	1	6
80–89	1	6

Differential Diagnosis.—In old-standing cases like those of Group I, Class C, we have to make the diagnosis from bone abscess, osteitis fibrosa, bone cyst, tuberculosis, central sarcoma and secondary carcinoma. In the other two classes of Group I the diagnosis is straight-forward for a radiogram gives unequivocal evidence. In Group II the exact diagnosis would be merely accidental.

On a radiogram bone abscess is localized in the cancellous tissue. Osteitis fibrosa and cystic disease are usually more widely spread. Tuberculosis is accompanied by more widely distributed osteoporosis and, in a lesion of this extent, would not be confined to a single tuberosity. In our cases the patella is normal and the femur, if involved, shows a healed fracture. Central sarcoma causes an expansion of the upper extremity. Secondary carcinoma should be accompanied by signs of the primary lesion.

The diagnosis is not difficult if the history, clinical signs and radiograms are carefully studied.

SUMMARY

1. Fracture of the upper extremity of the tibia is found in 1.2 per cent. of our skeletons.

2. It increases in frequency up to sixty years and most of our examples are found in skeletons between forty and sixty years. Thereafter, the percentage diminishes, probably because older people are less able to withstand the accident and its complications.

3. In 50 per cent. of our examples the lateral tuberosity is alone involved. In another 25 per cent. both tuberosities are injured. The remaining 25 per cent. includes fractures of medial tuberosity and intercondyloid eminence.

4. The fracture may be a relatively minor one, unrecognizable clinically

and registered anatomically by a mere crack in the articular surface. This occurs in 25 per cent. of our examples and forms our Group II.

5. Of more extensive fractures, Group I, there are three successive phases or classes. The first is a stave of the entire upper extremity on the shaft. The second is a comminuted fracture with splitting off, and in some instances impaction also of one tuberosity. The third is a depressed fracture of one condyle with disorganization of the tuberosity, but without stave of extremity or splitting of tuberosity.

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THE TREATMENT OF DIFFICULT FRACTURES OF BOTH BONES OF THE LEG

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Treatment of fractures of the shafts of the tibia and fibula is not infrequently difficult. Weight-bearing bones, which are of necessity surrounded by strong muscles, are subject to very definite distorting muscle pull. This distorting pull is stimulated by the fracture, which throws the muscles into spasm.

Fractures in the shafts of both bones of the leg are affected chiefly by the calf and peroneal groups of muscles, which, in addition to overriding, tend to cause very definitely an anterior angulation (calf group) and an internal angulation (peroneal group). The distorting pull of these muscle groups can be readily explained on the basis of their greater power and anatomical attachments.

Another definite factor which influences the treatment of weight-bearing bones is gravity, so that after muscular relaxation is obtained there is sagging of the fragments at the site of fractures. This holds true in fracture of both bones of the leg.

In the treatment of these fractures traction (skin and skeletal) and suspension by some type of splint are available. In the use of skin or skeletal traction there are several difficulties which may be encountered and which may be enumerated as follows:

(1) Distorting muscle pull with angulation and overriding which at times is difficult to overcome without adding a relatively heavy weight to the traction.

(2) Sagging of fragments is difficult to prevent by the usual method of suspension, such as with individual strips of cloth slung between the bars of the splint.

(3) Condition of the soft parts may be such that it is impossible to manipulate or apply skin traction.

The type of fracture plays an important part in reduction and the maintenance of that reduction.

To assist in overcoming these various difficulties the following method was devised by the author on the First Surgical Division at the New York Hospital.

This method makes use of skeletal traction by means of a Steinman pin through the os calcis, with fixation of the knee-joint by plaster-of-Paris splint at an angle to insure muscle relaxation, together with control of the other end of the fractured bones. It is as follows:

The patient is put under ether anæsthesia and a Steinman pin passed through the centre of the os calcis under the proper aseptic precautions. A sterile dressing is then applied over the pin. Following this a metal strap is attached to the pin, made up of two Parham bands passed through each other. (Fig. 1.) An assistant makes traction on the metal strap sufficient to overcome the overriding of the fracture. No actual attempt is made to manipulate the fragments. The knee-joint is then flexed to an angle of ninety degrees while sheet cotton is applied from the upper one-third of the thigh down to the Steinman pin. Extra padding is placed over the patella. Several layers of plaster of Paris are then placed on the extremity. At this time a wire spade is incorporated in the plaster. The spade extends up to the knee and well out beyond the foot, being in the same general horizontal plane as the tibia and fibula. Several more layers of plaster are applied,

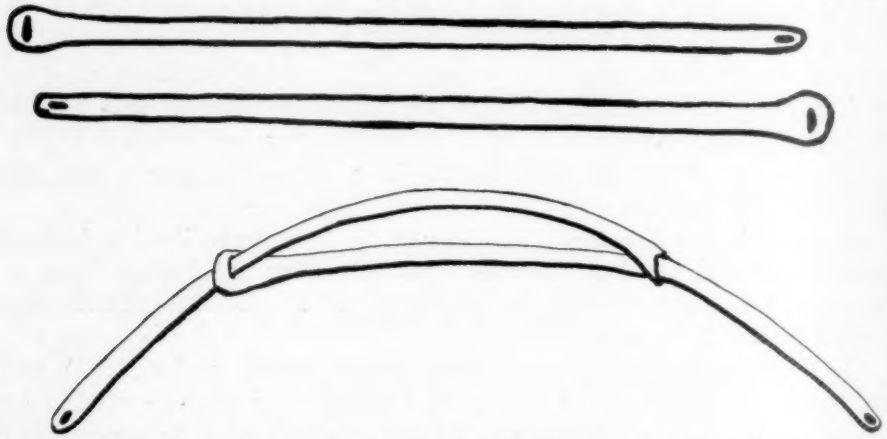

FIG. 1.—Diagram No. 1, showing two Parham bands passed through each other which are used as a stirrup on the Steinman pin.

sufficient to incorporate the wire spade and insure a firm splint. The plaster is allowed to harden and a pulley is attached to the end of the spade; a rope is attached to the pin and run through the pulley with a five-pound weight suspended from it. Weight may be increased or decreased or the pull may be changed in the lateral plane by simply shifting the pulley either side of the spade. Likewise, the direction of pull in the vertical plane may be shifted by placing the pulley below or above the end of the spade.

The patient is put to bed and the entire apparatus suspended from a Balkan frame. (Fig. 2.)

The leg is then X-rayed in twenty-four and forty-eight hours and again on the fifth day. Usually by the fifth day anatomical reduction has been accomplished. Then, with the traction still operating, cotton is placed about the foot and it is encased in plaster up to the original plaster. The slight gap between the two plaster splints is bridged by anterior and posterior moulded plaster splints which are in turn anchored to the circular splint by a few turns of plaster bandage. The fresh plaster is allowed to harden and in a few hours the Steinman pin is removed.

This gives a completed plaster-of-Paris spica extending from the upper one-third of the thigh down to and including the foot with the fractures reduced and held. (Fig. 3.) At the end of four weeks the plaster-of-Paris splint is bivalved, the fracture site tested, moulded plaster splints are then applied and the patient allowed up on crutches.

From necessity this method was devised to use on a patient, W. E., forty-seven years of age, who was admitted to the First Surgical Division, New York Hospital, May 28, 1926, with the history that ten days previous to admission he had fallen from a scaffold

FIG. 2.—Showing a plaster spica extending from upper thigh down to the ankle-joint with wire spade incorporated, and traction operating over the end of the wire spade. The entire apparatus is suspended from a Balkan frame.

and fractured both bones of the left leg. Manipulation and application of moulded splints was done. He was then admitted to the New York Hospital ten days after his accident with the original splints on. There was marked œdema of the foot and soft parts about the site of fracture. The skin over the lower one-half of the leg was covered by blebs. Under these blebs the tissues looked congested and angry. The fragments of the tibia were not in contact at all and there was internal angulation at the fracture site with outward displacement and external rotation of the distal fragments.

After admission an attempt at manipulation for better position resulted in a complete pulling off of the outer layers of skin over the lower one-half of the leg. Moulded plaster splints were again applied extending above the knee. Subsequent X-rays showed the position of the bone fragments not improved. Our röntgenologist reported a comminuted fracture of the left tibia at the junction of the lower and middle one-half, oblique fracture of the fibula at the same level, and in addition a second oblique fracture of the fibula up near its head.

Here was a case which could not be manipulated into position and due to the condition of the soft parts, skin traction or open operation was impossible.

The above apparatus was placed on this patient and a very satisfactory reduction was obtained in forty-eight hours. The pin was removed and the spica completed at the end of five days. The original plaster was removed at the end of four weeks, and firm union at the fracture sites was found. Moulded plaster splints were then applied, the patient was allowed home on crutches on the thirty-second post-operative day and reported to the hospital at regular intervals. After ten weeks his splints were removed and patient very gradually began to bear weight on his leg.

He was able to return to his usual occupation at the end of five months, with solid union, good weight-bearing line and no shortening.

FIG. 3.—Showing the plaster spica completed, including the foot, with the Steinman pin removed and wire spade still remaining.

Since this original case we have treated four other cases of bad fractures of both bones on the First Surgical Division, and one other case from the Second Surgical Division.

CASE II.—J. O., fifteen years of age, metal worker, was admitted to the New York Hospital, October 9, 1926, giving the history of having been struck across the left leg by a piece of iron weighing 400 pounds. X-rays showed a comminuted fracture of the left tibia, middle one-third, with an accompanying oblique fracture of the fibula with angulation and overriding. Manipulation and application of splints were attempted with poor results. Two days later our special apparatus was applied. An excellent reduction was obtained. The pin was removed in twelve days in this case and the patient allowed home on crutches wearing the moulded plaster splints on his twenty-first post-operative day. At a three months' follow-up he showed solid union and excellent weight-bearing alignment without shortening. He was then doing light work and later reported to me that he was doing the same heavy labor.

CASE III.—J. M., laborer, fifty years of age, was admitted to the First Surgical Division, New York Hospital, having been struck and his left leg run over by an auto-

mobile. X-rays showed a spiral comminuted fracture at the junction of the middle and upper one-third of the tibia with an accompanying fracture of the fibula. There was angulation, overriding and displacement of one of comminuted fragments. Manipulation was attempted with poor results. Our apparatus was applied two days later. The Steinman pin was removed on the twelfth post-operative day, while the original spica was removed at the end of three weeks. He was then allowed up on crutches with moulded plaster splints applied. He was allowed home on his fifty-ninth post-operative day, the delay being due to a complication of decubitus over the patella from insufficient padding. At the end of three months union was not solid, but seemed firm enough to allow him to wear a walking caliper brace. At the end of five months union was solid enough to walk without the caliper brace, and at my six months' follow-up he was walking normally with solid union, excellent weight bearing, and no shortening.

CASE IV.—P. E., female, forty-three years of age, was admitted to the First Surgical Division, New York Hospital, May 23, 1927, with the history of having been struck down by a taxicab which ran over her right leg. She had a badly compounded fracture of the right tibia which proved to be spiral in type at the junction of the lower and middle one-third. A débridement was done and a Lane plate applied to the tibia. Eleven days later, the patient's temperature having been high for some time, the wound was inspected and found to be infected. The Lane plate was removed and a long period of suppuration followed with a loss of considerable substance. In the meantime, due to dressing and handling of the leg, the fragments of both bones had been badly disarranged. Our apparatus was then applied one month following the removal of the Lane plate, with a window cut in the plaster for dressings. Following this, dressings and manipulations were far less painful to the patient. The fragments were found to pull into good alignment, especially the fibula. The pin was removed nineteen days after it was inserted and moulded splints applied, but of course there was no union due to loss of bone substance in the tibia. This patient continued coming to the Out-Patient Department for dressings and much to our surprise slowly got union, although the leg continued to suppurate. The fibula united first, which allowed the tibia to gradually fill in some of the gap which had been lost by suppuration.

At follow-up one year from her accident this patient has a small discharging sinus which is so slight that she does not come to the clinic for dressings at all. There is apparently solid union without false motion. There is some backward bowing at the fracture site in the tibia, but she is able to get about with the aid of a cane and a walking caliper brace.

CASE V.—F. S., male, thirty-nine years of age, was admitted to the First Surgical Division, New York Hospital, November 11, 1927, giving the history that just previous to admission, while directing the unloading of a barge of lumber, a large 12 x 12, 130 feet long beam, fell on his right leg, breaking it. X-rays showed a badly comminuted spiral fracture of the tibia at the junction of the upper and middle one-third with a comminuted fracture of the fibula at the same level. Manipulation was attempted with poor results. Three days later our special apparatus was applied. The pin was removed in nine days and about one week later moulded plaster splints were applied extending above the knee-joint. He was allowed home on crutches three weeks after his injury.

The patient continued to return to the Out-Patient Department and after eight weeks union was found to be firm, so that a walking caliper leg brace was applied. At six months' follow-up the patient was able to walk without any apparatus, union was solid, weight-bearing line excellent, and there was one-fourth inch of shortening in the right tibia. The patient was doing his usual work.

CASE VI.—A man, thirty-five years of age, was admitted to the Second Surgical Division, New York Hospital, with a fracture of the middle one-third of both bones of the right leg. He was treated in the same manner as the preceding cases, with a very satisfactory result.

I feel that these six cases represent as difficult types as one may encounter in the treatment of such fractures. The result in all except Case III has been very good and all of them have returned to their previous occupations, or could have returned, with solid union, good weight-bearing axis and without appreciable shortening.

Critical Comments.—(1) The plaster of Paris as applied fixes the knee-joint at an angle of ninety degrees, which puts and keeps the powerful distorting calf group of muscles at rest. It also acts as a constant means of suspension after muscular relaxation is obtained, so that it is impossible for the bones to sag at the site of fracture.

(2) Reduction is obtained by a minimum pull (five pounds is usually sufficient) due to complete muscular relaxation.

(3) The patient is comfortable at all times as there is no shifting of fragments to cause him pain. Likewise, with the suspension of the entire apparatus he is able to move about the bed freely.

(4) The Steinman pin can be taken out very early to avoid any bone necrosis or relaxation of the ankle-joint, while reduction is maintained by the completed plaster splint.

(5) Counter traction is distributed over a wide area chiefly against the posterior surface of the lower two-thirds of the thigh.

(6) With the incorporation of the wire spade, traction and counter traction are obtained on one piece of compact apparatus, so that a complicated set of pulleys and splints is not necessary.

(7) Patient is not able to adjust his own apparatus.

This method is published as one which has been used only in a limited number of cases, but in cases which are sufficiently difficult of management as to test its merits. It has come up to our best expectations, and being based on sound mechanical principles aimed toward overcoming the two chief difficulties in the treatment of fractures of weight-bearing bones (namely, distorting muscular action and gravity) it should continue to give good results.

For anyone desiring to use this method I would recommend fixation of the knee-joint at ninety degrees, with five- to eight-pounds' traction, removal of the Steinman pin and completion of the splint, on an average, after five days, with the removal of the original splint and application of moulded splints at the end of three to four weeks. Further treatment, such as allowing the patient a walking caliper leg brace, etc., should be carried out as indicated.

VALUE OF CLOSING COMPOUND FRACTURES BY SKIN PLASTIC

By John E. Cannaday, M.D.

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The advantages of early closure of a compound fracture are obvious. Many hospital days, suffering, deformities and lives are saved. Aseptic and antiseptic first aid methods are desirable. Our custom has been to apply a gauze pad, wet with tincture of iodine, mercurochrome, or picric acid solution. The limb is immobilized, the patient is sent to the hospital. If he is not in a state of shock the wound is carefully débrided and disinfected. This is done with a scrupulously careful technic, quite as much as if the intent were to open the abdominal cavity. After débriding the wound, removing grossly contused and contaminated tissue, and freshening the skin edges by paring off a narrow strip all the way around, the wound edges are sutured most carefully and should be very neatly and precisely approximated. Great pains are taken not to invert the edges, but rather to bring them together pointing upward, like the apex of an inverted "V".

Plastic Methods.—In event there is such a loss of substance or œdema that the edges cannot be brought together by usual methods, long, liberating incisions may be made some distance away, in order that the skin edges may be brought together over the exposed bone without undue tension. It is advisable, as a rule, that such liberating incisions be carried through the outer fascial covering of the muscles. We follow this method in order to provide for an improved circulation and to lessen the likelihood of long-standing œdema. If advisable, this work can be done under local anæsthesia. The patient is always given a prophylactic injection of antitetanic serum. The treatment outlined is routine with myself and my surgical associate, Dr. E. B. Henson, at the Charleston General Hospital. In more than one hundred cases of compound fracture treated by early skin closure, primary union was obtained in more than 90 per cent. of the cases.

The following illustrative cases are cited:

Case I.—A woman, sixty-eight years of age, in crossing a dusty road in midsummer, was knocked down by a car, receiving a compound fracture of the tibia and a simple fracture of the fibula. The tibia broke through the skin and protruded for at least two inches. She was taken to a nearby hospital and I was called to see her shortly thereafter. X-ray showed considerable comminution and overriding of the broken bones. Inasmuch as her general condition was fair, she was at once given ether anæsthesia and the wound disinfected as thoroughly as possible with fresh tincture of iodine, the badly lacerated and traumatized tissues were débrided, a portion of the grossly contaminated bone was pared away with a rongeur. The wound was carefully closed with silkworm-gut sutures. These were placed close together, not over one-fourth inch apart, and were allowed to remain in for approximately two weeks. Primary union took place. Bony union was delayed, but finally took place.

Case II.—A mine foreman was sent to me at the Charleston General Hospital. This man was unwell at the time of his injury, his trouble having been diagnosed as pulmonary tuberculosis of a chronic type. He had a bad productive cough and had had the same for many months. He suffered frequently from severe pains in his chest. He was the victim of a slate fall and, when looked over, was found to have a simple fracture of the left radius, one of the left femur, and a very bad compound fracture involving both tibia and fibula of the right leg. There was considerable destruction and loss of tissue over the tibia at the point of fracture. This man was

Fig. 1.—Compound Fracture of Tibia with Extensive Exposure of Bone at Point of Fracture.

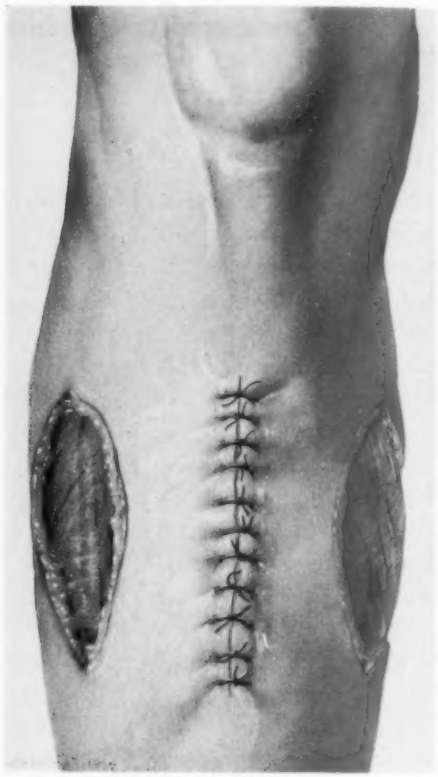

Fig. 2.—Showing Liberating Lateral Skin Incisions, Allowing Suture of Skin Wound Over Point of Fracture.

suffering great pain and was in a state of most profound shock. Cold perspiration, rapid, weak pulse, running 120 to 130 per minute. His general condition was so poor, even at that time, that his teeth were covered with sordes and his gums showed the presence of a severe form of pyorrhœa. He looked to be almost moribund. Splints were applied and the patient was put to bed. He was treated for shock. Glucose solution was given intravenously and hot water bags applied. Over the compound fracture was applied a dressing of gauze and cotton, wet with a two per cent. solution of mercurochrome.

The shock was severe and persistent. However, the next day the patient showed some improvement. At this time he was given 1500 units of antitetanic serum; still his condition continued poor. Three days later at noon, assisted by the resident physician on the traumatic service, I first débrided and then closed this compound fracture. The operation was done in the patient's room without moving him from his bed. The work was done under novocaine anæsthesia. Partly owing to loss of substance and partly

owing to swelling, large liberating incisions had to be made on either side of the leg. The fracture of the tibia was essentially a transverse one, and as the relative position of the ends was not good, it was improved by use of a bone skid. The wound caused by the original injury was snugly closed. This brought healthy skin over all of the exposed bone. The lateral liberating incisions on either side of the leg were approximately four inches in length and left a hiatus as much as an inch and a half in width at certain points.

For fear that these gaps in the skin might become grossly infected, they were treated with Carrel-Dakin solution for forty-eight hours. After that every three hours for a week. From this time on his granulating areas were dressed every other day with five per cent. balsam of peru in castor oil.

He stood the operation very well and had a fairly smooth convalescence. Most of the sutures were removed at the end of the second week. A few of them were allowed to remain for a few days longer for fear that there might not be firm union.

One month after injury this man's leg, so far as the skin is concerned, was so perfectly healed that it was hard to find the scar where his original injury was received. The liberating incisions on the side of the leg showed a smooth scar a little over one-fourth inch in width.

The leg is not swollen, the bony alignment is fair, and there is fibrous union. The man's chest condition has improved. Probably the rest in bed has helped that. At the end of the second month there was firm bony union.

CASE III.—Compound fracture of tibia and simple fracture of fibula; duration eight hours; wound disinfected, débrided and sutured, unfortunately under more tension than was desirable. Stay sutures were of silkworm gut placed quite close together. The site of the fracture had undergone partial healing so that healthy granulation tissue covered the bone; later, skin grafting was resorted to, the bony union was primary in spite of the fact that the skin did not hold. The protection afforded by the skin gave time for the building of a defensive wall at the seat of fracture.

CONCLUSIONS

After a rather extended experience with the methods above described we have found that the results are immeasurably better than when treated by the open method. The application of ordinary plastic methods serves to cover and protect the bone at the site of fracture and to prevent not only prolonged invalidism, but deformity and the formation of dense scar tissues directly over the bone. Such tissue breaks down easily and is always a menace.

THE TREATMENT OF FRACTURES OF THE CLAVICLE

A STUDY OF 422 CASES OBSERVED IN THE OUT-PATIENT DEPARTMENT OF THE ROOSEVELT HOSPITAL OF THE CITY OF NEW YORK

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Fracture of the clavicle is one of the most common fractures that comes to treatment, being second only to Colles's fracture in its incidence. So much has been written regarding its treatment that Kreisinger, writing in 1927, found descriptions of over two hundred devices for its treatment. Most of these come under the head of reducing dressings; *i.e.*, dressings designed to reduce the fragments or to hold them in alignment. Yet nearly all authors on the subject from Hippocrates to date admit that reduction is almost impossible to maintain and that a certain amount of deformity is to be expected. This deformity does not interfere with the function of the arm and tends to subside in time. With these facts in mind the value of complicated uncomfortable dressings is questionable. Certainly in cases of incomplete fracture, or fracture of the outer third without displacement, there can be no doubt that these dressings are more nuisance than they are worth, both to the surgeon and to the patient. The supporting dressing disregards the position of the fragments and merely aims to make the patient comfortable. It seems to be the logical dressing to use. To determine its value as compared with the reducing dressing a study of 422 cases treated in the Out-Patient Department of the Roosevelt Hospital between 1914 and 1927 was made. These cases were, of course, all ambulatory cases, and were treated by both reducing and supporting dressings.

A sling and binder or Velpeau bandage make no attempt at reducing the fracture or holding it reduced. They do support the weight of the arm and keep it from moving, thereby eliminating the two chief causes of pain and discomfort from the injury. Healing takes place as quickly with this form of dressing as with the others and the deformity is no greater as a study of the late results will show. It is, therefore, the dressing of choice in the great majority of cases.

Stimson, Scudder, Brewer, and other authors of text-books on fractures consider that a simple supporting dressing is sometimes the best form of treatment. Gibbon, quoting the observations made by Lucas-Championnière on jockeys, in whom fracture of the clavicle is a frequent injury, advises against any retentive dressing. He uses a simple figure-of-eight bandage between the shoulders with the arm supported by a sling and states that he has had excellent results when the sling alone was used. Colson, in reporting a large series of cases which occurred among children, advises the use of a simple sling entirely.

FIGS. 1, 2, and 3.—Method of application of sling. The tapes are passed through rubber drainage tubes so that they will not cut the skin of the patient. A body binder not depicted here is used with this type of dressing.

UNIV. OF MICH.

The sling is easily applied. It should extend beyond the elbow and be drawn snugly so that it will lift and support the arm. The ends should not be tied back of the neck, but rather crossed over the back, carried around under the axillæ and tied over the sternum. (Figs. 1, 2, and 3.) The tapes should be broad or else they should be well padded so as to prevent cutting the skin. A rubber drainage tube with the tape of the sling threaded through it makes a good protection. The weight of the arm is then borne by the shoulders and not by the back of the neck. A simple swathe around arm and thorax keeps the arm from moving except within narrow limits. After a week or ten days the swathe may be dispensed with, the patient wearing the dressing inside the clothes which act as sufficient binder. In from two weeks to eighteen days the sling may be worn outside the clothes and discarded entirely in from three to four weeks. When worn outside the clothes the sling may be knotted behind the neck.

While the arm is inside the clothes a Velpeau bandage may be used. Some surgeons object to its use on the grounds that it increases the deformity. It was used in forty-seven cases in this series but no untoward deformity was noted at the end of the treatment.

One case in this series may be quoted as an example of the value of the sling and binder.

The patient was a man of twenty-one years who was thrown over the handlebars of his bicycle, striking the pavement on his shoulder. He sustained a comminuted fracture of the middle third of his clavicle which might be considered a double fracture. There were two principal lines of fracture which separated a middle fragment about three-quarters of an inch long. All fragments were overriding and piled on top of one another, the inner fragment being lowest and the outer fragment uppermost. In view of the marked deformity the Dwight modification of the Sayre dressing was applied with considerable tension. The dressing caused so much discomfort that the patient cut it down almost as soon as he left the hospital. He returned next day with the arm loosely supported by the diagonal strip of the dressing. The same type of dressing was reapplied and cut down about as promptly by the patient. On his third return, next day, it was decided to make no attempt at further reduction although the deformity was still in its original state. Accordingly he was treated by a sling and binder. Union took place and he was discharged thirty days after his injury with good function of the arm but with marked deformity and three-quarters inch narrowing of the injured shoulder. He was seen four years later. At that time the deformity was slight and the measurement of the width of the shoulders showed the injured side to be no narrower than the other. Function was complete. By simply keeping the arm at rest the fracture had united and in the course of time the reparative processes of the body had so far overcome the deformity as to make it unnoticeable without close inspection. This is not an isolated case as the other late results show similar cases, although not so striking.

Even without treatment fracture of the clavicle will heal although the resulting deformity is apt to be greater. Three such cases came to the clinic after the union had taken place and were given no treatment. One case would not keep any appliance on and amounts to an untreated case.

The number of times the various dressings were used in this series can be seen by referring to Table I.

TABLE I.

Type of Dressing.

A. Cases Completing Treatment

Dressing	Complete	Incomplete	Comminuted
Dwight-Sayre	125	29	4
Sling and binder	19	15	2
Velpeau	25	23	
Figure-of-eight	8	6	
Cross	2		
Harness	1		

B. Cases Abandoning Treatment After Ten Days or More

Dwight-Sayre	30	4
Sling and binder	3	2
Velpeau	6	5

It will be seen that the Dwight modification of the Sayre dressing was the most popular. In considering this type of dressing it must be borne in mind that the patients were dispensary patients who were not seen more than twice a week and frequently only once a week. Hence, between dressings there was more or less slipping of the adhesive with consequent loss of the dressing's efficiency.

The average length of time required for the treatment was twenty-five days in the complete fractures and twenty days in the incomplete fractures. The dressing used seemed to make little difference in the time. The greatest time required was one hundred days. This was in the case of a man who applied to the hospital for treatment twelve weeks after his injury, during which time his arm had been kept immobilized by his local doctor. Another case in which the fracture was badly comminuted required three months. The remaining cases which needed more than six weeks to complete the treatment (eleven cases) had associated injuries to the shoulder which prolonged the treatment. Most of the cases needing more than a month received baking and massage. The others did not need it.

TABLE II.

Site of Fracture.

Outer third	65
Middle third *	250
Inner third	14
Not stated	93

Results.—In general it can be said that fracture of the clavicle will heal and give a good functional result. Even the four untreated cases had good function although the deformity was marked in three. The fourth was an incomplete fracture. So with the remainder of the cases in the series. Of

* Includes those of junction of middle and outer, and middle and inner thirds.

those cases which did not abandon treatment before four weeks there were only two with non-union.

One was in a man who remained under treatment eighty-four days. He had a four plus Wassermann reaction. At the time the last note was made on his record he had fibrous union and was getting antiluetic treatment. The other was in a girl of sixteen who abandoned treatment after thirty days. She also had fibrous union at the time. Attempts to trace both these cases have been unsuccessful.

TABLE III.

Type of Fracture.

Complete	241
Incomplete	112
Comminuted	7
Not stated	62

One hundred and eleven patients abandoned treatment before ten days and fifty abandoned treatment between ten days and three weeks. Such a large number of eloped cases is decidedly unusual in our other fracture cases and the reason for it is probably that they had no more symptoms rather than that they were displeased with the treatment. In many of our follow-up inquiries we came upon cases which had abandoned treatment because they thought they were all well and who had good results. One boy abandoned treatment after a week but returned a year later with a fracture of the other clavicle; he also abandoned treatment for the second fracture in a week and when seen in the follow-up had perfect function with the minimum of deformity. Undoubtedly some sought treatment elsewhere, either because they disliked us or because they lived too far away.

In the immediate results deformity is usual, especially in the complete fractures. Deformity also occurs in the incomplete fractures when there is angulation. In this series, however, marked deformity was noted in only fifty-three cases. Five of the seven comminuted fractures were discharged with marked deformity, one with moderate deformity and the other abandoned treatment. Three of the four untreated cases had marked deformity, the other being incomplete. Dressings designed to reduce the fracture as well as the simple supporting dressings were used. These cases are tabulated in Table IV. Apparently the deformity existing at the time treatment

TABLE IV.

Marked Deformity at Discharge.

Dressing	Complete	Incomplete	Comminuted
Dwight-Sayre	27	4	3
Sling and binder *	5	2	2
Velpeau	4	1	
No Treatment	3		
Not stated†	1		

* Two cases untreated for a week.

† One additional case in which type was not stated.

is instituted has more to do with the resulting deformity than the type of dressing used.

Late Results.—Follow-up inquiries were made only in those cases whose injury was subsequent to the summer of 1923. Clinic patients are inclined to be nomadic and usually move in less than five years leaving no address. There were sixty-one cases traced which were examined or reported by letter. The last case of the series was injured in February, 1927. Hence the cases in which the late results are given are all over twenty months old.

Function was complete in all of these cases, as would be expected from the immediate results. Two complained of a slight amount of pain at times but the pain was so slight that it did not interfere with the patients' activity. Thus from the point of view of function and comfort the results were nearly 100 per cent. perfect.

It is generally believed that the deformity resulting from a fracture of

TABLE V.
Cases without Late Deformity.

	Number of Cases	Type of Fracture			Dressing	
		Complete	Incomplete	Comminuted	Supporting	Reducing
Adults	11	8	1	2	7	4
Children	43	27*	13	0	29	14

the clavicle persists. The results in these cases indicate the opposite. Of the sixty-one cases only seven showed noticeable deformity. Those without deformity are from all classes; adults and children; complete, incomplete, and comminuted fractures. They were treated by supporting dressings as well as by dressings which strove to maintain reduction (Table V). Those who had late deformity have been tabulated individually. Four were in adults and three in children. In only three was the deformity marked. One of these

TABLE VI.
Cases with Late Deformity.

Age	Type of Fracture	Dressing	Deformity	Remarks
60	Complete	Sling	Moderate	Three-quarter inch shortening; deformity hidden by fat.
42	Complete	None	Marked	Would tolerate no dressing.
28	Complete	Sayre	Marked	
21	Comminuted	Sling	Slight	Three-quarter inch shortening at discharge; none in late result.
13	Complete	Figure-of-eight	Slight	
6	Incomplete	Sling	Marked	Incomplete fracture followed by refracture, also incomplete, two weeks after discharge.
5	Complete	Sayre	Slight	

* Three cases in which type of fracture was not stated.

was an untreated case. One was in a refracture through the previous line of fracture, both fracture and refracture being incomplete. The other case was treated by a dressing whose aim is to maintain reduction. These figures show forcibly that uncomfortable and intricate dressings designed to hold the fragments in alignment (whether they do or do not) are of no more value than a simple, comfortable, supporting dressing.

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THE TREATMENT OF SUPRACONDYLAR FRACTURE OF THE HUMERUS

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In 1910 Ashhurst published a monograph on fracture about the Elbow. It marked a great change in the prognosis of fractures, which, to that time, were treated with the hope that the elbow would be restored to normal, but with the expectation that it would not. The secret of his success in the treatment of supracondylar fracture of the humerus rests upon two propositions: accurate reduction, and fixation in hyperflexion. The improved results obtained since that time by men following these principles have proved his teaching correct.

From an anatomical and clinical study of this fracture the following conclusions were drawn:

1. The carrying angle will be preserved if reduction is complete and the forearm is accurately superimposed in hyperflexion upon the humerus before the arm is brought across the chest.

2. The triceps is the only muscle vitally concerned in the reduction of the fragments; its pull must be overcome before alignment is possible; it will hold the reduced fragments in apposition when the elbow is hyperflexed.

3. "Volkman's Ischæmic Palsy" will occur less frequently when complete reduction has been effected.

Uncomplicated supracondylar fracture with the most common type of displacement, the lower fragment backward and upward and the upper fragment resting upon the anterior capsule of the elbow, will be considered. Assuming that the diagnosis has been made and confirmed röntgenologically, replacement should follow at once. Unless there is marked hæmorrhage in the tissues swelling should not prevent immediate reduction. General anæsthetic is desirable. To watch fluoroscopically the fragments return to their proper position is very reassuring.

Two movements are essential: traction and hyperflexion. The assistant holds the humerus while the surgeon grasps the wrist with one hand and the lower fragment with the other hand, placing his fingers around the condyles and his thumb against the lower end of the upper fragment. With steady, firm traction on the extended forearm, and backward pressure with the thumb upon the upper fragment, the fractured surfaces are guided into alignment. If the lower fragment will not come into position it is because enough traction is not made. Having apposed the surfaces, the forearm is hyperflexed and accurately superimposed upon the humerus. Hyperflexion is defined by Ashhurst as that degree of flexion which can be obtained without shutting off the radial pulse. Because a valgus deformity at the elbow

is less deforming than a varus, some prefer to overcorrect by bringing the little finger in line with the great tuberosity of the humerus.

The forearm is then dressed in the flexed position and rotated medially across the chest. In hyperflexion the fragments are firmly locked and cannot be thrown out of position. If the forearm is brought across the chest before hyperflexion is complete the lower fragment will tend to rotate upon the upper, to separate laterally and override medially. Healing in this position will result in a valgus or gunstock deformity. (Fig. 1.)

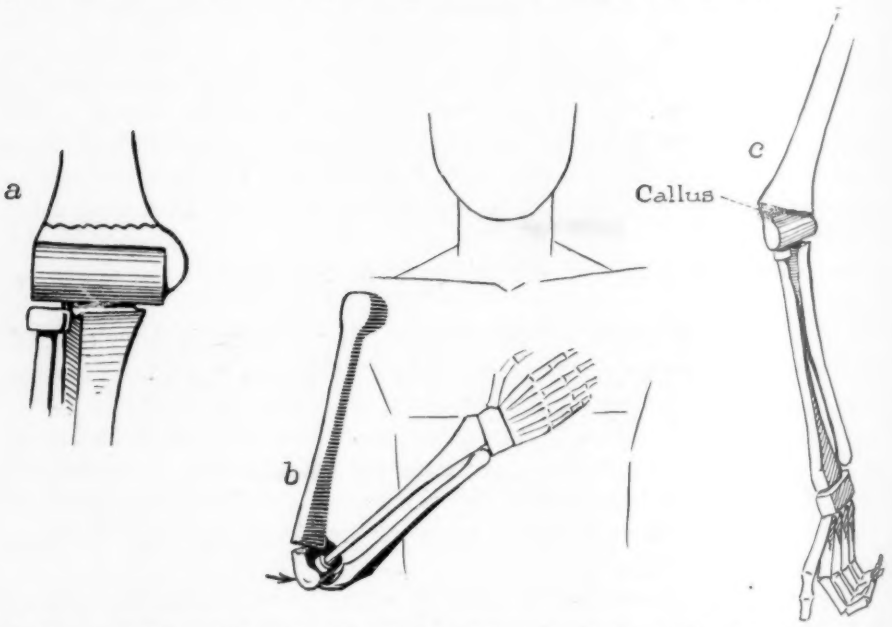

FIG. 1.—*a*—Schematic drawing of the elbow to illustrate mechanics. *b*—Medial overriding, lateral separation or rotation of the fragments upon each other as a result of bringing the forearm across the chest before the fragments are locked in hyperflexion. *c*—Healing in this position resulting in a valgus or gun-stock deformity.

Complete reduction allows the forearm to be flexed to an acute angle without using force. The space between the humerus and the forearm is greater when the lower fragment is brought sufficiently far anterior. (Fig. 2.) In most cases there will be ample room in hyperflexion for the soft tissues and the swelling incidental to the injury without danger to the blood supply. Vascular disturbance (Volkman's Ischæmic Palsy) is apt to follow when the forearm with the lower fragment improperly reduced is forced into position against the resistance of diminished space.

There is in our museum of pathology the amputated forearm of a boy who suffered a supracondylar fracture of the humerus. The arm had been set in acute hyperflexion and the boy sent home. When the doctor called the following morning the arm and hand were black and lifeless. If the patient cannot be under competent observation following the fixation of the fracture in hyperflexion, it will be safer to place the arm in a right angle position. It is only fair to state that some competent surgeons advocate

FIG. 2A.—Anatomical specimen of an artificially produced supracondylar fracture with triceps and joint capsule intact. Note the increased space in the elbow in a properly reduced fracture (indicated by line).

FIG. 2B.—Improperly reduced fracture and diminished space for soft tissues.

FIG. 3A.—In hyperflexion the triceps is tense and the fragments are interlocked.

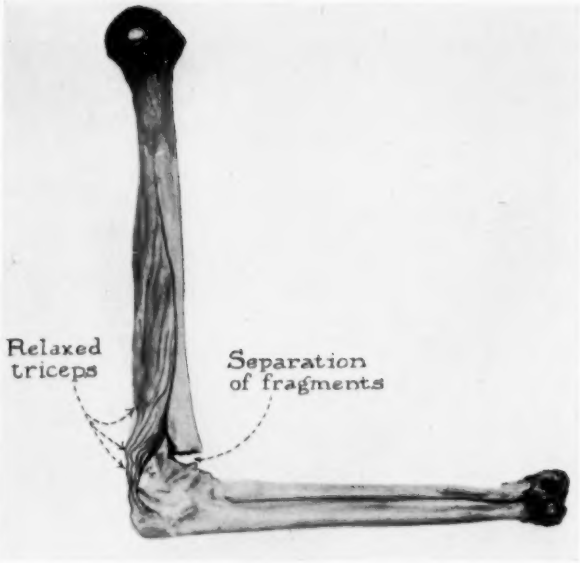

FIG. 3B.—At a right angle the triceps, in an anatomical specimen, is relaxed; in the living, is partially contracted and tends to separate the fragments, pulling the lower upward and backward.

right angle fixation as a general rule, and report good results. We advocate hyperflexion as a rule; but certainly partial limitation of motion with a valgus or varus deformity is nothing compared with the loss of an arm or an arm crippled by vascular disturbances as a result of over-zealous hyperflexion.

The triceps muscle, inserted into the tip of the olecranon, contracts and draws the lower fragment upward and backward. Before reduction can be executed its pull must be overcome by traction. In the position of hyperflexion (Fig. 3 *A*) it becomes tense and holds the fragments interlocked. It also prevents slipping when tense as a firm posterior moulded splint. The danger of dressing these fractures at a right angle is apparent. (Fig. 3 *B*.) In this position the surfaces are not firmly apposed because the muscle is not tense, and the direction of force is not to appose, but rather to separate the fragments. This is especially true because the line of fracture in most instances runs backward and upward.

It makes little difference how the properly reduced fracture is held in hyperflexion. Ashhurst advises a simple bandage. Scudder suggests adhesive fixation. Lund devised an ingenious bandage about the body. We follow the teaching of Kellogg Speed and use a posterior moulded plaster splint. This is comfortable and holds the arm firmly where it is placed. It has the added advantage of holding the hand in supination when this is desired.

The routine post-operative care of the average supracondylar fracture is made simple. The child is kept in the hospital for twenty-four hours under very careful observation. The dressing is adjusted if necessary, and the patient dismissed with the instruction to return bi-weekly. The dressing is removed each time, the arm inspected and powdered, and the patient asked to move the arm as much as he can. This will usually be not more than five to ten degrees. At the end of two weeks the splint is bent to a right angle and the arm lowered to that position. Upon the next two visits it is made certain that hyperflexion is possible. The posterior right angle splint is replaced. At the end of the third week the splint is discarded and the arm supported in a sling. All dressings are removed at the end of the fourth week, and instructions are given the patient or its parents to soak the arm in hot water twice a day and to move the arm actively. If complete extension is not possible at the end of the fifth week weight carrying is begun. Carrying a small pail in which a little more sand is put each day stimulates the interest and coöperation of the child.

TRANSACTIONS

OF THE

NEW YORK SURGICAL SOCIETY

STATED MEETING HELD NOVEMBER 28, 1928

The President, Dr. Frank S. Mathews, in the Chair

TROPHIC ULCERS OF THE FOOT

Dr. Abraham Hyman presented a boy, fifteen years of age, who was admitted to Mount Sinai Hospital November 24, 1926. He had first noticed several blisters on the sole of his left foot some four months previously, which he had attributed to narrow shoes. Infection of these blisters had followed, causing swelling and pain in the foot. Two months later the swollen area had ruptured about two centimetres below the small toe. He entered the Flushing Hospital, where for seven weeks conservative treatment, consisting chiefly in the application of wet dressings and baking of the foot, was carried out without improvement. Two weeks after his discharge from that hospital he developed a suppurating sinus at the site of the ulcer, and was then sent to Mount Sinai Hospital.

At Mount Sinai he gave a further history of pain in the left leg, spine and shoulders for three months, with a loss of fifteen pounds in weight. For the two days prior to admission there had been tenderness in the inguinal glands on the right side. With the exception of the above, the past history was irrelevant, the only two items of importance being a tonsillectomy and appendectomy respectively two and one-half years previous to the present illness.

He was a poorly nourished boy, with teeth discolored and pyorrhœic. On the plantar aspect of the left foot, at the head of the fifth metatarsal bone, a small, irregular opening in the skin presented, moderately tender and with necrotic tissue at its base. There was no evidence of inflammation around this area. In the corresponding region of the right foot a portion of the skin about the size of a twenty-five-cent piece was found to be thickened and hard, having the appearance of a callus. In the centre of this area was a small opening about one millimetre by two millimetres in size, and the region around this was markedly tender without, however, showing any sign of inflammation. The patient's temperature on admission was 102, but dropped to normal in a few days. Examination of the urine was negative.

X-ray of the feet revealed a thickening of the periosteum of the left fifth metatarsal with no evidence of bone destruction. The picture gave the impression of a partial dislocation of the metaphalangeal joint of this toe. Neurological examination was negative except for a right facial asymmetry. There were no sensory disturbances. His condition improved under treatment so that he was discharged from the hospital December 3, 1926.

Three weeks after his discharge he was again admitted to the hospital, having in the interim been under treatment in the dispensary. He now complained of swelling of the left foot, with pain more marked at night than by day. There was constant discharge from the sinus on the left foot and from an ulcer which had formed at the site of the callus on the right foot. On examination a red ulcerated area about the size of a twenty-five-cent piece over the head of the fifth metatarsal of the left foot was disclosed, with some

induration at the periphery and tenderness all around this spot. X-ray of the spine was negative; that of the left foot showed a periosteal thickening of the fifth metatarsal similar to the condition previously noted, with an area of bone destruction at the head.

Operation.—December 28 the little toe of the left foot was amputated at the metatarsophalangeal joint. Pathological examination of the specimen removed showed acute inflammation of the soft parts with fibrous atrophy of the bone. Neurological findings failed to demonstrate any signs pointing to syringomyelia or any other organic disease of the central nervous system.

When he left the hospital on January 13 there was a small, discharging sinus which at the time he was next seen in the return clinic, February 2, was closed. May 16 an X-ray of the feet showed no abnormality of the remainder of the metatarsal bones. There was, however, an irregular area of destruction on the upper anterior surface of the os calcis which gave the appearance of a small, localized osteomyelitis.

July 7, 1927, he was readmitted to Mount Sinai Hospital complaining of swelling and pain in the left foot. The big toe was reddened in appearance, but not tender. On the plantar surface of the proximal phalanx of the great toe a third punched-out ulcer about one centimetre in diameter had formed, which was discharging pus. A small scar presented on the lateral surface of the foot, near the point of removal of the fifth toe. His general condition was unchanged. There was good pulsation in the dorsalis pedis on both sides. Typhoid vaccine injections were given intravenously, the dosage July 7 being five millions, which was doubled on the 15th. The local condition seemed to improve under this treatment, and a third inoculation of twenty-five millions was given on the 22nd, followed by a fourth of forty millions on the 24th of the month. Subsequent to these inoculations the ulcer of the toe healed entirely.

July 21, at the time of his discharge from the hospital, an X-ray of the left foot showed rarefaction at the head of the second and third metatarsal bones. The Wassermann was negative, as was the urinalysis; the blood count was normal.

After leaving the hospital he was well for a time with the exception of a few attacks of pain in both feet, associated with fever, chills, vomiting, and headaches, and pain in the muscles of the lower extremities occasionally. March 9, 1928, he was admitted for a fourth time complaining of almost constant pain in the feet. The ulcers, which had healed over under the typhoid vaccine, had reopened of late. The general physical condition remained unchanged.

The ulcers on the plantar surface of both large toes were each about the size of a twenty-five-cent piece, and there was a smaller ulcerated area in the region of the small toe of the right foot. No neurological signs of sufficient importance to warrant a diagnosis of disease of the central nervous system could be obtained. There was pulsation in the large vessels of both feet. The oscillometer showed equal and normal circulation on both sides.

Typhoid vaccine was again administered intravenously, with the result that the ulcer on the left foot healed in a short time, and that on the right foot was almost healed following the last injection of one hundred and fifty millions. Examination of the throat and nasal sinuses was negative. Dental examination revealed a few infected teeth, which were extracted at once. He was discharged from the hospital April 2, 1928.

The patient was next seen April 18, at the time of his fifth admission to the hospital. Following extraction of the infected teeth he had developed a submaxillary adenitis. Examination of the feet showed the ulcers almost

healed, there being only a slight discharge from a superficial ulceration on the left foot. The Quickenstedt test showed some evidence of a partial block in the spinal canal. Both the neurological surgeon and the dermatologist on consultation diagnosed the condition as trophic ulcers of the feet.

July 5 of this year he was admitted to Mount Sinai Hospital for a sixth time. On examination the condition of the feet was found unchanged. The ulcers had reopened, there being two on the plantar surface of the left foot and one in a corresponding site of the right foot. He also complained of dull headaches, without, however, any blurring of vision. X-ray of the right foot taken July 14 showed a periosteal thickening of the entire shaft of the fifth metatarsal, with deformity of the head.

July 17 a left peri-arterial sympathectomy was done. The femoral artery was exposed in Scarpa's triangle. A large inflammatory gland was found overlying the artery, and this was excised. The femoral artery was denuded for a distance of about two inches above and below the entrance of the profunda. On the second post-operative day it was noted that the left foot felt warmer than the right, and the callus on the sole of the foot was less painful and softer, both of which conditions obtained in an increasing degree on the fourth day. Aside from a slight infection of the wound which cleared up with wet dressings there were no complications.

For a time there was improvement in the local condition of the left foot, the ulcers becoming more and more superficial; but a few weeks after leaving the hospital the ulcers reopened and the condition reverted to that previous to operation. August 25 he again entered the hospital because of lymphangitis of the left leg, which cleared up under wet dressings. The status at the last examination, November 23, was as follows: One ulcer on the sole of the right foot; two on the sole of the left foot; slight pain experienced on walking.

This case presents a young man with trophic ulcers of both feet yet with no demonstrable neurologic or vascular organic disease to account for the condition. X-rays have on a number of occasions showed changes suggestive of Köhler's disease; but in the descriptions of that disease the reporter had been unable to find any mention of associated ulcers. In going over the X-rays of the spine it is to be noted that there is only a partial fusion of the fifth lumbar vertebra. The radiographers seem to attach little importance to this defect, which may, however, be of some significance.

Dr. Walter M. Brickner called attention to the fact that the röntgenogram of the spine in Doctor Hyman's case showed a cleft in the arch of the fifth lumbar vertebra. To be sure such a cleft in the fifth lumbar, somewhat less often in the fourth lumbar or the first sacral arch, is a very common finding; in many cases it is, apparently, without any clinical significance. Nevertheless it is sometimes the only local objective finding in cases with symptoms of spina bifida occulta. In his published study of that disease (*American Journal of the Medical Sciences,* April, 1918, vol. clv) Doctor Brickner had said that we must recognize four groups: (1) Those with external signs (hypertrichosis, congenital lipoma, telangiectasis), with symptoms; (2) those with external signs, without symptoms. (3) Those without external signs, with symptoms. (4) Those without external signs, without symptoms. Doctor Hyman's case may very well be one of spina bifida occulta of the third group. Certainly, the recurring trophic ulcers with callous borders,

as presented and described, are quite like those seen with spina bifida. The failure, of all attempted therapy, and the apparent hopelessness of the condition, justify, in Doctor Brickner's opinion, an exploratory operation on the spine. In his series none of the cases operated on for spina bifida occulta suffered any ill effect. To be sure, such operations are not likely to be followed by brilliant results since there is usually an irremediable lesion in the cord. Sometimes, however, the release of caudal filaments adherent to the dural sac effects relief, and this might happen in Doctor Hyman's case. In one of the cases of spina bifida occulta operated on by Doctor Brickner the release of adherent caudal elements in the dural hernia was followed by healing of frequently recurring ulcers of the buttocks. In the course of subsequent years trophic ulcers and gangrene involved several of the toes, but ulcers on the buttocks never appeared again.

ANURIA COMPLICATING FRACTURES OF THE EXTREMITIES

Dr. Abraham Hyman presented a boy, ten years of age, who was admitted to Mount Sinai Hospital, March 25, 1927, with a history of an automobile injury a few hours previously. Examination revealed a fracture of both femora in the middle third with displacement of the distal fragments mesially. The left elbow region showed a fracture through the lower end of the humerus with displacement outward of the lower fragment and, also, fracture of the internal condyle. Under open ether anæsthesia the fractures of the femora were reduced under fluoroscopic control, and a large plaster-of-Paris case applied. A röntgenogram taken the following day demonstrated that there was still some displacement of the fragments; the case was removed accordingly and on March 30 another case applied. For this purpose a second anæsthetic was administered. Both thighs were put up in abduction with partial flexion of the knee-joints. The elbow was put up in flexion.

From the time of his admission, March 25, until April 4 the patient passed normal amounts of urine which showed a faint trace of albumen, with a specific gravity of 1012; on one occasion a few granular casts were found. On April 4 there were two attacks of macroscopic hematuria. An examination of the fundi at this time showed them normal; the blood pressure was 150/100. April 5 the patient stopped voiding, and catheterization yielded only ten cubic centimetres of bloody urine. The next day the urinary output ceased entirely, vomiting ensued, the pulse became rapid and of very high tension, and the blood chemistry examination showed an urea of 67, nitrogen 113, uric acid 8.6, and creatin 3.1. The few drops of urine obtained were loaded with red blood cells, occasional white blood cells, and hyaline and granular casts.

Despite medication and diuresis there was absolutely no urinary output, and on the third day of his anuria, in view of the high blood chemistry and the clinical evidences of uræmia, it was decided to do a decapsulation. Under gas and oxygen anæsthesia both kidneys were exposed. The kidneys were not engorged or glaucomatous; if anything, they were paler than a normal kidney. Both kidneys were decapsulated and a small section of kidney parenchyma removed for histological examination.

The morning after the operation a blood chemistry examination showed that the figures had mounted, the urea being 82, nitrogen 131, uric acid 10, and creatin 3.4, with a carbon dioxide of 53. Within twenty-four hours following the operation the patient voided five ounces of urine. The next day the urinary output increased to twenty-four ounces and there was profuse

urinary drainage from both lumbar incisions which had been drained. The histological report on the specimen of kidney parenchyma removed was "slight tubular degeneration with cloudy swelling".

The urinary output steadily increased to forty-two ounces at the end of the third post-operative day, the nausea disappeared and the vomiting ceased. The blood pressure dropped from 150/100 to 115/65, and the blood chemistry figures gradually returned to normal. The wounds healed kindly, the urinary leakage ceasing within ten days. From this time on the convalescence was uneventful. The urinary examination now shows a normal specimen.

ECTOPIC SUPERNUMERARY URETER OPENING INTO THE VAGINA

Dr. Abraham Hyman presented a girl, sixteen years of age, who had since early childhood constant dribbling of urine day and night. There has never been a period when this has ceased. She voids normally four or five times during the day. Her general health has always been excellent. She has been examined on a number of occasions, and treated for a nervous form of enuresis. She was a well-developed, healthy girl. There is considerable excoriation of the labia and the inner aspect of the thighs, the result of constant dribbling of urine. A first inspection of the vaginal outlet was negative. A catheter was then passed into the bladder and some eight ounces of clear urine withdrawn. A pledget of cotton was then placed in the vaginal orifice; about ten minutes later this pledget was soaked. The bladder was then filled with an indigocarmin solution, and another pledget inserted and removed ten minutes later. It was found soaked with urine, but colorless, thus demonstrating that it was not a vesicovaginal fistula. By means of a strong light and a speculum, a tiny opening was revealed on the anterior vaginal wall, just behind the external meatus. Drops of urine were seen coming from this orifice. Attempts to probe the orifice with fine catheters or bougies were negative, an obstruction being encountered at about one centimetre distance. The condition was apparently an ectopic ureter opening into the vagina. A cystoscopic examination showed a normal bladder and ureteral orifices. Both ureters were catheterized to pelvis and clear urine was obtained, with good equal function as determined by indigocarmin. The urine from the third ureter contained no blue after thirty minutes, and was of a pale, watery consistency. The catheterized specimens from the two normally placed ureters contained 1.2 per cent. urea; that from the ectopic ureter showed 0.2 per cent. urea. A röntgenogram of the urinary tract was negative. A film taken with two opaque catheters *in situ* showed a normal position of both kidneys. The ureteral orifice in the vagina was then injected with iodide. After about thirty cubic centimetres had been introduced the patient complained of pain in the left loin. A röntgenogram then taken showed a considerably dilated ureter, ending in a small pelvis in the region of the left kidney. A pyelogram of the left pelvis showed a normal outline, and was in close proximity to the pelvis communicating with the third ureter.

Operation.—September, 1927. A left lumbar incision was made, exposing a normal-sized kidney with an accessory ureter coming off the upper pole of the kidney. This ureter was markedly dilated all the way down to the pelvis. The normal pelvis was situated in the middle of the kidney and was not dilated. A careful examination of the blood vessels was made and it was found that the vessels entered the kidney in their normal relation, with the exception of a large branch of the renal vein which entered the upper pole of the kidney behind the ureter.

Procedure.—It was decided to resect the upper pole of the kidney. The ureter was opened, and a finger introduced into the pelvis to demarcate its

outline. The upper pole of the kidney was then resected, after ligating the branch of the renal vein above-mentioned. The ureter was freed as far down in the pelvis as could be reached through a lumbar incision, cut across, and the stump carbolized. The upper pole of the kidney was closed with mattress sutures, using fat implants, and the wound closed in layer sutures, with rubber dam drain.

The dribbling stopped immediately after operation. The patient made an uneventful convalescence and was discharged from the hospital within three weeks after operation. A control cystoscopy on November 14 (about two months after operation) showed that indigocarmin appeared in equal concentration from both kidneys within a few minutes after intravenous injection, and that there were no evidences of retention of urine in the left kidney. There has been no incontinence at any time since the operation.

BILATERAL DOUBLE PELVES AND DOUBLE URETERS

DR. ABRAHAM HYMAN presented a man, twenty-eight years of age, who was admitted to the hospital in January, 1922. He had been complaining of pain in the back and frequent urination for two years. These symptoms had been increasing in severity of late, and the patient had noticed that his urine was turbid. Neither kidney was palpable or tender. The röntgenogram of the genito-urinary tract was negative. The urine was cloudy, containing large amounts of pus. The phenolsulphonephthalein test was 40 per cent., blood chemistry normal; the Wassermann negative. Cystoscopic examination showed an inflamed bladder. The right ureteral orifice was considerably retracted, and an impassable obstruction encountered at fifteen centimetres. No flow of urine or indigocarmin was obtained from this side. The left ureteral orifice, which was normal in appearance, was catheterized and clear urine obtained, with good indigocarmin within fifteen minutes. At the time the first cystoscopy was done, it was not noticed that there were two extra ureteral orifices. A diagnosis of right sided infected hydronephrosis was made.

At operation it was discovered that there was a double pelvis with two ureters. The kidney was found atrophic and hydronephrotic. In view of the fact that there was practically no kidney tissue left, a nephro-ureterectomy was done, removing the kidney and ureters in one piece down to within an inch of the bladder. This necessitated an anterior extra-peritoneal incision. After operation the patient's urine was perfectly clear for about a week, when it suddenly became turbid. In view of the findings on the right side, a similar condition was suspected on the left.

The patient did not return again until October, 1927. In the interval he had been fairly well except for attacks of pain in the left lumbar region with moderate frequency of urination. The urine had been increasing in turbidity and the patient finally consented to undergo a second cystoscopic examination. This showed an inflamed bladder. There were two ureteral orifices seen on the right side. These could only be catheterized a short distance. The ureteral orifice on the left side was considerably swollen. Above and to the outer side of the ureter there was an inflamed, œdematous area which appeared to be a second ureteral orifice. This orifice, however, could not be catheterized, and no indigocarmin was observed coming down. The normally placed left ureteral orifice was catheterized to the pelvis and clear urine obtained, with strong indigocarmin. The catheterized specimens showed occasional pus cells. At a third cystoscopy the same findings were observed, it being impossible to catheterize the second orifice. A diagnosis

was made of double kidney and double ureters, with infection of the lower pelvis and ureter.

Operation.—October 14, 1927. A left lumbar incision exposed a double kidney, the upper half represented by normal, solid looking parenchyma. The lower half was hydronephrotic. The kidney was somewhat contracted in the middle at the point of fusion of the upper and lower halves. The ureter coming from the upper half of the kidney was normal in size and appearance; that from the hydronephrotic part of the kidney was considerably dilated. Fortunately, the main blood supply entered the upper half of the kidney. A heminephrectomy was done, with removal of the dilated ureter as far down as could be reached through the lumbar incision. The bleeding was controlled by means of mattress sutures.

The day following operation the patient passed only a few ounces of urine, and for the succeeding twenty-four hours the output was about four ounces. The patient then became stuporous, although he could be aroused. The blood chemistry, which prior to operation showed an urea of twenty-three, now rose to thirty-two. The urinary output steadily increased, under diuretics. This peculiar stupor lasted for about three days and then entirely cleared up, the condition evidently being a post-operative psychosis. Despite the fact that his urinary output increased steadily, his blood chemistry mounted, the urea reaching as high as seventy-three milligrams a few weeks after operation. A considerable collection of pus was found in the wound. After thorough drainage his general condition gradually improved and the urea dropped down to forty-nine milligrams.

His condition now is very good. The urine is perfectly clear, and a blood chemistry taken a few weeks ago was normal.

POLYPOID GASTRITIS—PARTIAL GASTRECTOMY

Dr. Edward Rutherford Cunniffe presented a woman, thirty-one years of age. Fifteen years ago she was operated upon for redundant colon for which a colopexy was performed. The appendix was removed at that time. She had a tonsillectomy done five years ago. She had enjoyed good health since that time.

In July, 1928, she contracted a sore throat and was confined to bed for one day. The glands on both sides of her neck were painful, tender and swollen. She improved slightly during the next few days and returned to her home four days after illness began, despite continued prostration and fever. Three days later she began to vomit. She was unable to retain anything she ate or drank. Vomitus was sometime bile-stained, but at no time was there any evidence of blood. There was no pain at this time except that associated with vomiting. She remained at home for three weeks. Her condition did not improve and pain and tenderness appeared in upper abdomen. The reporter saw her first August 11, 1928. Her temperature was 101° F., pulse 110, respiration 24. Tenderness was indefinite over her entire upper abdomen. She was admitted to the Union Hospital on that day, where she remained for two weeks; during that time temperature remained at 102° F., for first four days of her stay, pulse dropped from 100 to 80 and respiration about 20. White blood cells were 18,000, 72 per cent. polymorphonuclear leucocytes. After four days her temperature gradually became normal. Chief complaints at that time were persistent vomiting, distention and generalized abdominal pain, which at times localized in the right upper quadrant and epigastrium. X-ray examination revealed an apparently normal gall-bladder—duodenum and pylorus negative. Her progress was slow and vomiting was difficult to control

during the first week of her stay. She then improved somewhat, and was discharged, August 14, 1928, still vomiting and very nervous.

She was not seen again until October 9, 1928, when she returned complaining of an aggravation of all her symptoms of abdominal pain, tenderness and persistent vomiting. She had been unable to retain even fluids during the past ten days. Another gastro-intestinal series taken during this time showed a constant large defect in the pars pylorica. She was again admitted to the hospital, and on October 9, 1928, under gas, oxygen and ether anæsthesia, the abdomen was opened by incision to the right of the median line. Many adhesions were encountered, due no doubt to the previous operation of colopexy. When the stomach was brought up into view, the pylorus was about twice the normal size and felt quite soft and thick, and was of a peculiar consistency which was typical of neither a carcinoma nor an ulcer, but resembled somewhat a leather-bottle stomach. This condition extended approximately fifteen centimetres along the stomach and was more marked over the greater curvature, but could be felt at the lesser curvature near the pylorus. A partial gastrectomy was then done.

During the first post-operative day the patient vomited three times small amounts of dark red fluid, which was of no particular consequence. During second post-operative day she vomited one ounce of light colored fluid and did not vomit again. During first three days after operation she was supplied with fluids, intravenously, subcutaneously and by Murphy drip. Glucose was given with insulin. From that time her recovery was smooth and uneventful. The Murphy button passed by rectum on the nineteenth post-operative day. Has had but one attack of vomiting since operation, this occurring four days ago when she thought she was guilty of over-eating.

Specimen.—Upon examining the excised portion of the stomach a very thickened and polypoid condition of the mucous membrane was found both in the upper part of the section and at the pylorus. A distinct polypous outgrowth was found in the upper part of the specimen and another large one at the pyloric ring.

Beginning just proximal to the pylorus and for a distance of seven centimetres the mucous membrane was entirely missing and appeared as if a cast of the mucous membrane had been removed, denuding the entire circumference of the stomach.

Pathological Report.—Specimen consists of a resected portion of stomach pylorus and four centimetres of duodenum. The mucosa is raised and thickened. For a distance of four centimetres over pyloric valve, the mucosa is completely denuded down to muscularis, which is covered with a diphtheritic membrane. There is an ulcerated sac one centimetre deep beneath the mucosa on the lesser curvature.

Microscopical.—In all of fourteen sections taken from various portions of the specimen the condition was found to be polypoid gastritis with no signs of malignant features.

A later report from Doctor Ewing, received November 26, 1928, is as follows: "I would prefer the diagnosis of chronic gastritis with atrophy of mucosa, and a very marked lymphoid infiltration and hyperplasia with ulceration. Only one polyp seen that is not connected with the ulcer. True polypoid gastritis shows adenomatoid polyps. In this case the elevations are due to lymphoid hyperplasia."

RECURRING INTUSSUSCEPTION

Dr. Edward R. Cunniffe presented a boy, first observed during July, 1927. He was then six years of age and complained that for the previous six months he had suffered with recurring attacks of general abdominal pain and vomiting. The pain would occur very suddenly and was always followed

by vomiting (with slight periods of remission of pain). This condition would last for about twenty-four hours; they would occur about twice every two weeks, with frequent vomiting. There was never any elevation of temperature and no blood was found in the stools. An exploratory laparotomy was advised, to which the parents did not consent at that time. They returned on September 8 with a history of four similar attacks since last examination, two months before.

The boy was operated upon at Union Hospital September 10, 1927. When the abdomen was opened a small, easily reducible intussusception was found in the ileum. This reduction was accomplished by simply picking up the ileum when the intussusception reduced itself. The appendix was removed. On further examination large mesenteric glands were found in the region of the lower ileum. The abdomen was closed without drainage and convalescence was uneventful. The patient was discharged from the hospital on the fourteenth post-operative day. The boy was not seen again until February 26, 1928, when he returned with the history that he had had attacks of abdominal pain and vomiting similar to those previous to his operation. The first one came on one month after leaving hospital and since that time had recurred every two or three weeks and lasted for about twenty-four hours. He had an attack two days before which, during the first twenty-four hours, was similar to his previous attacks, but his condition steadily grew worse during the second twenty-four hours, with frequent vomiting and abdominal pain. The patient was acutely ill, and had a pinched facies, and complained of severe generalized abdominal pain. His temperature was 102° F., pulse 130, respirations 26 at this time. Abdominal examination revealed some resistance, but no definite rigidity. A large, tender mass could be felt in the right lower quadrant. On rectal examination no mass could be palpated, but some blood was noticed on examining finger. The patient was immediately sent to the hospital and the abdomen was again opened at the site of the old scar. The mass was easily brought up into the wound and found to be an irreducible intussusception of ileum into ileum, about ten inches from cæcum.

The involved portion was very dark in color, and a slight attempt at reduction caused a rupture of the wall. No further manipulation was done. On account of the desperate condition of the patient, the mesenteric vessels were ligated and the intussusception was excised. The free ends were brought through the abdominal wound and sutured as in a Mikulicz operation, except that a tube was sutured into each lumen, as recommended by Paul. No Paul's tubes being available, plain rubber tubing was used. Patient drained freely of intestinal contents, but condition was very poor during first twelve hours with vomiting of black foul-smelling fluid. Infusion saline 600 cubic centimetres given, Murphy drip by rectum. Patient's condition continued poor. That night patient was restless, but draining freely, and no vomiting; temperature was 106° F., pulse 140, respiration 30.

The drainage of intestinal canal through tubes was profuse, but it was apparently a very important factor for after the first twelve hours the condition improved very rapidly. However, the tube slipped out of one end of intestine and although replaced it could not be held in intestine. There was also some necrosis of the protruding portion of the bowel, and the skin about the wound became very much excoriated by the discharge.

Various means were tried to improve the condition of the skin so that a suture anastomosis might be done, but it was difficult and little improvement was noticed. April 10, an attempt was made to crush the spur, as in a typical Mikulicz operation. A heavy clamp was placed on the spur and

tightened as much as possible; leaving it on until the clamp was found free. Although this was tried with various clamps at different intervals they were unable to break down the spur. A special clamp was devised with which they were now successful and the patient began to pass some fæces by rectum. However, the fistulous opening was so large that there was still considerable drainage from this opening. After waiting until June 14 to secure a better external condition for closing the intestinal gap and finding little improvement, the small intestine was mobilized (under ether anæsthesia) and the opening closed as is sometimes necessary in a Mikulicz operation on the large intestine. This was very successful, no leakage occurred. Patient had a normal stool forty-eight hours later and thereafter his convalescence was uneventful.

Doctor Cunniffe remarked that this boy illustrated the recurring type of ileal intussusception without satisfactory etiology, no tumor being found in this specimen. No other pathology was found in the abdomen except several enlarged mesenteric glands. He called attention to the method of treatment for providing drainage of the intestinal tract.

THROMBOSIS, INFARCTION AND EMBOLISM

Dr. Charles F. Farr read a paper with the above title, being a clinical and statistical study from the First Surgical Division (Cornell) of the New York Hospital, for which see page 481.

Dr. Lewis A. Conner remarked that the old and long controversy as to whether thrombosis was the primary condition or whether a change in the surface of the intima—in other words phlebitis—was the primary process in thrombosis is still unsettled. Doubtless the condition is not always the same. The last ten or fifteen years have brought to light a great many interesting facts and there are many resemblances between thrombosis formation in the veins, and the formation of sand bars in flowing streams. The resemblances are very close in some respects, but the sand-bar analogy cannot be applied too strictly. Nevertheless thromboses do form, as do sand bars, where there are eddies in the stream at the junction of veins, or where there is slowing of the current due to widening just above the vein valves. These are the places where thrombosis usually begins. In considering the etiology of these post-operative cases one has to bear in mind these facts. Doctor Conner was inclined to think that improvement of the venous return is a much more important consideration than any low-grade infection or extension from the wound. It is very difficult to understand why, for example, in a perfectly clean operation on chronic appendicitis thrombosis can develop in the left femoral or popliteal region. One of the points worth emphasis is that a venous thrombosis forms very gradually. There is a deposit of blood platelets at the point where the thrombosis begins and this often seems to be a very gradual process, a matter of perhaps a week or two before the vein is completely occluded by the thrombus. That period, when the thrombus is forming and when the vein is not yet occluded by the thrombus and the blood is still flowing in it, is the time when minor emboli are very apt to occur, even before there is any local clinical signs of

thrombosis. One should make a sharp distinction between these small emboli, which are apt to be released early in the process of thrombosis, and the massive, fatal emboli which result from dislocation of a large, fully formed thrombus. These latter seem to be much more frequently seen in post-operative states than in medical conditions.

The symptoms resulting from the small emboli are often mild and may easily be overlooked, especially if local signs of thrombosis have not yet appeared. A sharp attack of pleural pain, with or without a cough, and some blood-streaked sputum may be the only symptoms. While it is not always easy to distinguish between these milder attacks of pulmonary embolism and post-operative inflammatory conditions in the lung, the differential diagnosis can usually be made. The attacks of embolism are apt to occur later in convalescence (usually in the second week), to begin with sharp, pleural pain and to have no cough or fever until a day or two later. Post-operative pneumonia, on the other hand, usually gives symptoms during the first two or three days after operation and fever and cough usually precede the pleural pain, if indeed any pain be present. Moreover, when blood shows in the sputum in cases of embolism it is frank, unchanged blood, either bright or dark, and has not the characteristic, diffuse rust color of pneumonic sputum.

In the attempt to prevent the development of thrombosis after operations a great variety of measures had been suggested and employed. Acting upon the theory that the chief predisposing cause was the slowing of the blood stream in the large veins, the Germans had for some years urged that patients after abdominal operations be gotten out of bed at the earliest possible moment. It was the speaker's impression that statistics had thus far failed to show any important lessening of the incidence of fatal pulmonary embolism as a result of this practice. Lockhart-Mummery had urged the avoidance of preliminary purgation and of a starvation diet before and after operation, the furnishing of suitable mattresses for the operating table, the avoidance during operations of such strained positions as may cause pressure on the veins of the extremities, and the encouragement of free movement of the limbs after operation. It seemed reasonable to suppose that tight abdominal bandages, which restrict greatly the free movement of the diaphragm and so interfere with one of the important factors in assisting venous return from the lower part of the body, might predispose to thrombus formation and should therefore be avoided as far as possible.

Dr. Seward Erdman understood Doctor Farr to say that in none of the fatal cases of embolism had there been any symptoms recognized even in retrospect which suggested development of a thrombus.

Many surgeons will agree with Doctor Farr as they call to mind some tragic experience with a fatal pulmonary embolus, occurring on the tenth or eleventh day after a simple, clean herniotomy, and an apparently smooth convalescence.

The speaker had gone in detail over the charts of several such cases and

did find in each instance some slight, but perhaps very significant persistence of temperature into the third, fourth or fifth post-operative day.

If such charts were compared with the average perfectly normal post-operative temperature curves the discrepancy would be apparent.

Furthermore, in review of the charts, in several instances there was mention in the nurse's bedside note that "patient complained of pain in the leg", which must have been so slight that no attention was paid to it.

As to length of time which intervened between the onset of the acute signs of pulmonary embolism and the actual death of the patient, this in most cases was probably not more than fifteen to thirty minutes, or even less. Occasionally such cases linger on much longer, even up to four or six hours and it is interesting to speculate on the possible explanation.

About five years ago, on the Second Surgical Division of the New York Hospital, a woman died of pulmonary embolism in the course of a smooth post-operative convalescence. About half an hour after the onset of the desperate embolus symptoms she complained suddenly of pain, numbness and tingling in both legs and lost control of them. She lingered on for about two hours after the onset. At autopsy the usual findings of a large clot blocking the pulmonary arteries was found, but there was also the very unusual finding of an eight-inch-long snake-like clot in the abdominal aorta blocking the bifurcation into the common iliacs.

This clot was of similar appearance to that found in the pulmonary artery and suggested a similar point of origin.

The heart was now carefully examined and the foramen ovale was found to have a rather wide slit-like opening from right to left auricle, large enough, it was felt, to have allowed the clot arriving in the right auricle to pass into the left auricle and thence to the aorta.

The anatomists, Quain and Gray, state that "not infrequently" a small, slit-like opening persists throughout life, at the upper edge of the foramen ovale; perhaps 20 to 30 per cent.

Doctor Erdman believed that when an embolus blocks the pulmonary artery, the pressure on the right side of the heart must rise above that on the left, and in this manner would tend to force venous blood from the right auricle over into the left auricle, if any opening existed through the auricular septum.

This, of course, would be a reversion toward the fœtal heart circulation.

This theory would be the plausible explanation of the clot found in the abdominal aorta at autopsy; but, furthermore, may explain the prolonged period of symptoms in certain cases of fatal pulmonary embolism.

Dr. Richard Lewisohn pointed out that in about five cases recorded in the German and Scandinavian literature, the pulmonary embolus was removed and the patients were cured. It is difficult to say whether any of these cases might have recovered automatically.

Dr. John Douglas said that apparently Doctor Farr has found very

little which would appear in the way of etiological factors beyond infections. The discussion of the last speaker and also that of Doctor Erdman made him want to ask Doctor Farr if in these fatal cases there was a sufficient length of time that any of them lived, which would have made possible operative interference. There must have been a very small number where the patient lived long enough to do an operation of that kind and at the same time not give the impression that they would get well anyway. One hesitates to risk life by removing clots from the pulmonary vein if there seems a prospect of spontaneous recovery. Another question in these cases of pulmonary infarction from small emboli, which are apparently occurring where one could possibly reach them by operation, has Doctor Farr any evidence or presumptive evidence that it would be of any use to try to remove the foci from which the emboli arise in these cases? Doctor Douglas did not mean where it comes from appendicitis. He had one post-appendix case last summer that had pulmonary embolism occurring about the eighth or ninth day. He had another patient, a woman, who, while under observation previous to operation, developed a pain in the chest and the next day complained of pain in the leg and gradually developed more or less migrating superficial phlebitis in the internal saphenous vein. Is there anything to be gained in these cases by tying the saphenous vein?

Doctor Farr, closing the discussion, stated he had seen cases in which a clot in the right heart had progressed into the pulmonary artery and apparently had been the cause of death, and in which there had been no other thrombosis.

On studying the charts of these fatal cases one frequently, perhaps always, would find some evidence that things were not progressing normally. On the other hand, in his series there had not been one case, so far as he knew, in which the actual condition of thrombosis had been determined before death. During the last three weeks he had had three post-operative cases of phlebitis of a decidedly definite type—the usual superficial phlebitis following various abdominal operations. These as usual had ended in recovery and he had learned to expect good results in all such obvious cases of phlebitis.

As to getting ahead of the process of phlebitis by excising the vein he had once done this in a case from the Medical Division. The patient suffered from enteric fever with severe infection of the internal saphenous vein. Many abscesses had formed along the course of the vein. The vein was ligated proximal to any palpable thickening and the abscesses were opened separately. The patient recovered. Certainly in the great majority of obvious cases excision or ligation is uncalled for while the very nature of a deep and hidden phlebitis would preclude diagnosis and operative treatment.

TRANSACTIONS

OF THE

PHILADELPHIA ACADEMY OF SURGERY

STATED MEETING HELD JANUARY 8, 1929

The President, Dr. Astley P. C. Ashhurst, in the Chair
Calvin M. Smith, Jr., M.D., Recorder

STAB WOUND OF THE LEFT VENTRICLE

Dr. William Bates presented a man, twenty-eight years of age, who was admitted to the Accident Ward of the Graduate Hospital November 15, 1928. He had an incised wound in the anterior aspect of the left chest. The wound in the chest wall was about one and a half inches long, was vertical and just to the outer side of the left border of the sternum. The patient's temperature was normal; his pulse forty-eight. The heart sounds showed a curious scraping or catching in the rhythm. The pulse rate quickly mounted and the area of dulness in the precordial region rapidly increased. The wound was explored by enlarging the vertical incision upward and downward. It was found that the knife had passed through the costochondral junction of the third and fourth left ribs. This detachment was utilized for further exploration and the intercostal muscles between the second and third and between the fourth and fifth ribs were severed and the ribs broken about two inches from their traumatic detachment. This was made possible by a skin incision at right angles to the middle of the original incision.

This osteoplastic flap was laid back and an opening in the pericardium exposed to sight. Bleeding was rather steady and came from within the pericardium rather than from a cut vessel in the edges of the pericardium. The pericardial opening was enlarged upward and downward. At first nothing could be seen because of the bleeding, but gradually by sucking and sponging it was seen that the left ventricle had a wound in the right upper portion. It was impossible to determine whether or not its opening communicated with the left ventricular chamber, but from the amount of bleeding the reporter believed that it did not. The muscular bleeding was very steady and with each contracture and rotation of the heart there was a steady spray of blood carried in the form of an arc on the table drapings above the wound. The wound in the ventricle was closed with the chromic catgut. The pericardium was then washed out with normal saline and the pericardium was being closed when the heart stopped. A little finger irritation through the wound and ten minims of one to one thousand adrenalin chloride intravenously was given. Intravenous normal salt solution was then started. The pericardium was about closed when the heart again stopped, but gentle massage and additional adrenalin in the intravenous solution reëstablished contractions and no further cardiac arrest occurred. The osteoplastic flap was brought into place after the pericardium was completely closed. A small wick drain was put down through the intercostal space on top of the pericardium and the skin was closed with interrupted silk sutures.

During the operation the pulse rate went to 108. The patient was in the hospital twenty-seven days. His leucocytes immediately following opera-

tion were 10,200 per cubic millimetre, and just prior to discharge were 10,400. The Wassermann was strongly positive; the blood chemistry during his stay in the hospital was normal. The cardiac dulness was greatly increased to the left, and X-ray of the heart showed a very decided increase of the cardiothoracic ratio. Both by electrocardiogram and X-ray this widening steadily decreased until the last X-ray showed a practically normal relationship. Five days after the operation the electrocardiogram showed a true picture of acute coronary occlusion. One week later, or twelve days after operation, with a pulse rate of 90, the electrocardiogram showed no evidence of coronary occlusion and Dr. James M. Talley stated that chronic coronary occlusion might develop later. Two weeks later, or twenty days after operation, with a pulse rate of 75, the electrocardiographic tracing again suggested coronary occlusion. A fourth reading, taken just prior to discharge on December 12, showed no coronary occlusion. During that part of his stay in the hospital when the temperature was elevated and the patient had a high leucocytosis, it seemed as if it were going to be necessary to drain the pericardium for fear of pyogenic invasion of the hæmopericardium. This, however, was not necessary and the patient now is able to do some work while continuing to receive rather strenuous antiluetic treatment.

MORTALITY OF INTESTINAL OBSTRUCTION

Dr. Selling Brill (by invitation) read a paper with the above title for which see page 541.

Dr. John B. Deaver said, regarding strangulated hernia, that in his experience most cases of strangulated hernia have had too much taxis before being referred for operation as demonstrated by blood in the sac and in the mesentery, the latter particularly favoring gangrene. When it was his privilege to teach undergraduates he always told them not to do taxis, but to operate immediately.

In operating for strangulated hernia, a mistake that often occurs is when having opened the sac and before releasing the strangulation not to cleanse it, as well as to have smears and cultures taken of the fluid contents. Where this precaution is not taken and the fluid contents are infected, release of the strangulation makes communication with the peritoneal cavity, and through this communication the infected fluid can find its way into the abdomen and result in infection of the peritoneum. Intestinal obstruction following operation for appendicitis can usually be avoided. In the speaker's experience the majority of these obstructions occur in the lower abdominal abscess cases. Quite a percentage are the result of the involvement of the terminal coil of the ileum when it forms a part of the wall of the abscess. Where he finds the terminal ileum involved to the extent that it has lost its flexibility, he makes an ileocecostomy or an ileocolostomy. This prevents both immediate and remote obstruction. As this is his practice, he rarely sees an obstruction any more. Bear in mind that this ileocecostomy or ileocolostomy, as the case may be, is not done with the object of arresting peritonitis, if present.

Dr. George P. Muller said that we were apt to be carried off our feet by the many beautiful pieces of work being done; that of Hayden and Orr

being the last of a long series, in which the whole thing is made to appear as a form of toxæmia which can be combated by replacing the electrolytes. In experimental lower intestinal obstruction—the kind we usually see—there was no disturbance in the electrolytes, so one cannot expect to do a great deal of good by pumping salt solution into the patient. Doctor Brill found that mortality was directly traceable to delay in operation, in all types of cases except a few congenital herniæ, etc. In the herniæ there was no mortality in the first twelve hours, but after that it was 50 per cent. Regarding paralytic ileus, this occurs mostly in patients with general peritonitis, *i.e.*, when the abdomen was opened it was found swimming with pus. These patients made a desperate fight for a few days and then increasing distention occurred. As to enterostomy Doctor Muller believes that it rarely accomplishes anything except drainage of the loop selected. It is only when peristalsis is very active—and it rarely is—that the upper intestine can be emptied. With Doctor Deaver, the speaker does not believe in jejunostomy; it does no good except in the rare cases where one can demonstrate peristalsis and reverse it. In obstruction following appendiceal operations, when we close with some angulation of the lower coils, it means trouble in a week or ten days. This is traceable to the effects of operation. However, in patients where the appendix had been stripped out of a gangrenous area which reached down into the mesentery of the ileum and obstruction occurs later it is hardly any fault of the operator. In order to avoid obstruction the speaker adopted the McBurney incision, as in this way one gets drainage outside of the coils of the ileum, and no drainage across the coils. It definitely lowers the percentage of obstruction following operation. The speaker thinks the Deaver operation of ileocolostomy is entirely too severe for the average patient. He would not feel justified in making a mid-line incision to do an ileocolostomy in the presence of acute peritonitis.

Dr. Damon B. Pfeiffer said that he has seen a few cases where he is sure that the performance of jejunostomy saved life. In the last few months the speaker had such a case in the Presbyterian Hospital, in which instance jejunostomy certainly saved life. There was infection of the lower abdomen following resection of carcinoma of the cæcum. The infection was limited to the right lower quadrant. The patient became greatly distended and for six days never passed gas; his condition was such that he was practically in collapse and looked ready to die at any moment. In these circumstances jejunostomy was made and he drained off two or three gallons of fluid by the next day. No gas was passed by bowel until three days later. This patient gradually recovered. Doctor Pfeiffer has had two or three other similar cases and believes that there is a small definite field for jejunostomy in which it is definitely a life-saving procedure. He did not wish to stress the indications for its use, but simply to ask for an open mind on the subject.

Dr. Charles F. Nassau said that one need not be afraid to give morphin, particularly in the presence of peritonitis. The bowels should

be put in splints after operation. An endeavor to make the patient's bowels move is dangerous; it is wiser to almost forget that they need to move. In abscess cases, having achieved localization by the Ochsner treatment, why spoil the effect by creating peristalsis which is dangerous and sometimes fatal in the presence of adhesions? He has had two patients who were doing well when the over-anxious attending physicians ordered castor oil because the bowels had not moved for two or three days and who immediately developed obstructions. Until the patient is practically healed up one should depend on the use of enemas for bowel movements.

Sometimes the surgeon treats a strangulated hernia, particularly of the femoral type, where lack of experience will cause him to do an intestinal resection. In this way a certain number of patients are lost, even if the operation be done under local anæsthesia. A little hope and faith in the ability of a bad looking piece of bowel to recover itself when put back where it belongs, the abdomen drained by a tube of gauze and rubber dam to the bottom of the pelvis and enough gauze in the opening to keep the bowel back, will bring much better results. In a number of cases the speaker has put back bowel that years earlier he would unhesitatingly have resected. In other words, one can afford to take the chance of having the formation of a fistula. He is in accord with Doctor Brill in his remarks about fancy operations after the original work. If the original work be well done, let the patients alone and they have a better chance for recovery. Emptying one loop of bowel fifteen feet from where the damage is will not do the patient a particle of good.

PERFORATED PEPTIC ULCER

Dr. John B. Deaver read a paper with the above title for which see page 529.

Dr. Charles F. Nassau said that if, instead of division of the pyloric sphincter, which is perfect as far as it goes, the surgeon would do a Finney operation, he would accomplish the same thing; it is a perfectly safe procedure; it cures; and the patients almost never have recurrence of their ulcers. Doctor Nassau does not use clamps, having lost one patient whom he believes died of a necrosis following the use of a clamp.

Dr. George P. Muller said that years ago he read a paper by Judd suggesting the performance of the Ramstead operation. He has done it five or six times since then and has had one case of marked pyloric stenosis without evidence of ulcer in which that operation was done in either 1920 or 1921. The woman is well and was relieved of her symptoms. This is a minor procedure as compared to that which Doctor Deaver performs. The speaker did not quite see how Doctor Nassau reasons that the Finney operation will give no mortality when compared with gastro-enterostomy. It is easier to do a gastro-enterostomy than to do a Finney operation, and that should in a way make for complications. The Finney is a difficult operation to do even with practice; there is always a tendency for the lower

edge of the duodenum to tear away. Apparently no operation on the stomach is one hundred per cent. successful and one has to decide in the individual case. He has been doing the Judd operation, taking out an oval section and thus getting a wide-open pyloric end; it is infinitely easier to do. Some years ago, while in Rochester, the speaker saw Judd do twelve of these operations in a few days, and was exceedingly impressed with certain facts, namely, that he had not been making large enough incisions and not making the oval sufficiently big. So far no one operation has proven always successful.

Dr. John B. Deaver said that he could concur in a great deal, but not with all that has been said. He thinks that Finney can accomplish more with his operation than anyone else can. The speaker has done it and had good results, but with Doctor Muller he thinks it more of an operation than is posterior gastroenterostomy. Marginal ulcer will occur after a Finney operation. He agrees with Doctor Muller that there is not any one procedure which is absolutely perfect.

BRIEF COMMUNICATIONS

A METHOD FOR THE PERFORMANCE OF ENTEROSTOMY IN CASES OF STRANGULATED INGUINAL HERNIA WITH GANGRENE OF THE BOWEL

My observation is that it is the strangulated form of hernia that is usually most poorly handled. There are certain handicaps and serious obstacles to be overcome here. The operator usually has no means of knowing the proximal from the distal end of the bowel. It would be hazardous and unwise and a waste of time for him to prolong the operation sufficiently to find out, when he does not know, for instance, by the nearby location of the cæcum, or some such landmark. Enterostomy drainage can take care of both ends if used in accordance with the method described. If the bowel is damaged, but partially viable, necrotic spots should be reënforced by suture. Inasmuch as the damaged segment of bowel is likely to be the seat of a mechanical obstruction, a suitable enterostomy should be done. The intrabowel portion of the tube is carried all the way through the damaged loop, the tube having previously been split spirally and several lateral openings made in its sides with scissors. The tube so arranged will drain from healthy bowel at both ends of the damaged segment.

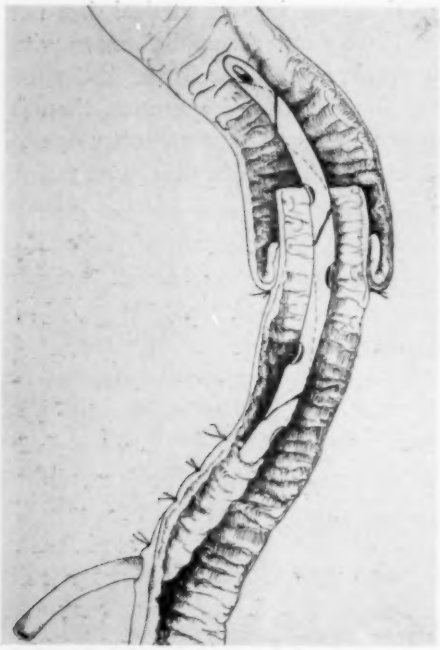

Fig. 1.—Enterostomy in Strangulated Inguinal Hernia, with Gangrene of the Bowel.

Believing that the enterostomy opening should be protected and made as valve-like as possible, I have not only made use of the Horsley method of securing it in the bowel, but have, when available, tucked omentum about it by suture, or passed it through a perforation in the omentum. In case the omentum is absent, however, I have found that the appendices epiploicæ of the sigmoid made a most satisfactory substitute.

As to reaction of necrotic bowel, an end-to-end anastomosis by the invagination method is by far the speediest type of anastomosis; also one of the safest as regards the possibility of leakage, always safeguarding the anastomosis by an enterostomy.

My observation has been that a considerable number of lives have been lost by dilly-dallying with makeshift methods in dealing with bowel necrosis,

when discovered in the hernial sac. To secure results the surgeon should act with decision, accuracy and despatch. Such methods as leaving both ends of the necrotic bowel in the hernial wound with a drain in each with the hope or the idea of anastomosis at a later date may be of possible value in certain cases, but I believe that, generally speaking, a definite resection with anastomosis as previously described will give by far the lower mortality.

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FIBROSARCOMA DEVELOPING IN A DESMOID*

STEWART and Mouat and others[3, 6, 7] have studied desmoid tumors. The former reviewed sixty-six cases of fibromas (desmoid tumors) of the abdominal wall. They found that these neoplasms occur most frequently in women who have borne children and that they commonly arise from the musculo-aponeurotic structures of the anterior abdominal wall below the umbilicus. Morison and Drummond observed that these new growths in the abdominal wall are rarely tender on palpation. With the patient prone, raising the head or shoulders does not cause the tumor to disappear but sometimes makes it more apparent. Early in their growth the tumors enlarge parallel to the muscle-fibres; later they may encroach laterally. Balfour reported seven cases of desmoid tumors of the abdominal wall, two of these developing in close relationship to an abdominal incision. Nichols found in thirty-one cases of desmoid tumor that twenty-five of the tumors occurred in the abdominal wall and nineteen apparently arose from the aponeurosis of the rectus muscle.

FIG. 1.—Section from margin of tumor illustrating fibrous connective-tissue components. In lower half are a few muscle bundles engulfed by new growth. (x 75.)

There is frequently a recurrence of the tumor after incomplete removal. Morison and Drummond reported a case in which there was a history of four operations, three of them for recurrence. Several observers[1, 4, 8, 9, 11] have mentioned the possibility of malignant change. Stewart and Mouat, on the other hand, in their series did not find evidence of sarcomatous metamorphosis.

The structure of a desmoid tumor, according to Ewing, "usually varies from that of a hard acellular fibroma to a rather cellular fibrosarcoma, the latter exhibiting active growth and not infrequently recurring after incomplete removal. The prognosis is good and there seems to be no record of the development of definite malignant qualities or metastasis. The majority of these tumors respond slowly but satisfactorily to radiation."

* Submitted for publication July 6, 1928.

REPORT OF CASE.—An unmarried woman, a bookkeeper, aged twenty-two, came to the clinic because of a painless, symptomless tumor of the anterior abdominal wall below the umbilicus. She had discovered the lesion accidentally. The tumor had increased slightly in size in the two months between its discovery and examination at the clinic.

Examination revealed a hard fixed tumor apparently involving the lower part of the left rectus abdominis muscle. A clinical diagnosis of desmoid tumor of the abdominal wall was made. At operation, a hard gray to white fibrous tumor was removed. The new growth apparently began near the space of Retzius and extended upward in the substance of the left rectus abdominis muscle for about 9 cm. Grossly the tumor was composed of interlacing whorls of gray to white fibrous tissue and scattered between these were light brown to tan softer areas. Microscopic examination of tissue from the fibrous areas of the tumor demonstrated the usual fibrous connective tissue-cells in the irregular arrangement so common in desmoid tumors. In certain areas of the tumor the muscle-fibres were engulfed by the new growth. Sections of tissue from the light brown to tan areas were much more cellular. The cells were distinctly fibroblastic in character and there were numerous mitotic figures (Figs. 1 and 2). A pathologic diagnosis was made of a fibrosarcoma in a desmoid tumor.

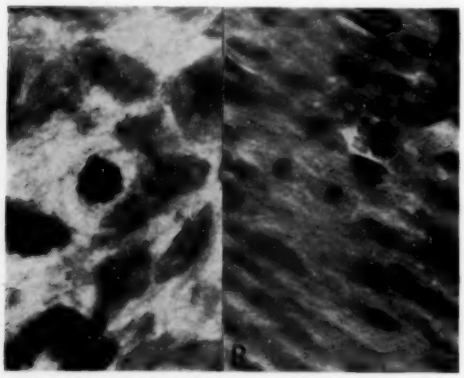

FIG. 2.—Mitotic figures from a more actively growing portion of the tumor. *A* (x 800) and *B* (x 350).

Eighteen hundred milligram hours of radium treatment was given over the abdominal wound. Five months after operation the patient was apparently in good health.

Several points of especial interest are associated with the case reported. The patient had not borne children and laparotomy had not been performed, which is contrary to the common rule in these cases. The presence of sarcoma in the desmoid tumor is very unusual. At the time of the operation malignant change was suspected in the new growth and the suspicion was borne out by our subsequent microscopic examination.

Mitotic figures in neoplasm are strong evidence of malignant change. Most of us, if asked to give the microscopic signs of malignancy, mention the presence of mitotic figures in cells among the first of the changes. In the diagnosis of tumors arising from connective tissue the significance of irregular mitosis is undeniable. MacCarty, however, believes that mitotic figures are not necessary for the diagnosis of malignancy in cases of carcinoma, and Broders believes that they are not necessary in the diagnosis of epithelioma. In certain tissues, as for example, the germinal centres of lymph nodes, tonsils, or the epithelium of the cervix, mitotic figures are not infrequently found; of themselves, they are not indicative of malignant change.

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HÆMANGIOMA OF CHEST WALL WITH UNUSUAL SYMPTOMS *

THE patient, a woman aged twenty, was admitted to the Surgical Service of St. Luke's Hospital, New York, October 25, 1927, with the following history:

Three years ago without known cause a soft painless swelling suddenly appeared in the second right intercostal space close to the sternum. At first this swelling was only noticed when she bent forward or on standing erect; on lying down it disappeared completely. During the last year and a half the swelling has been growing larger and does not now completely disappear on lying down. In addition a small hard nodule has appeared in the swelling. The nodule is tender and at times cannot be found, at other times when caught it will suddenly slip from between the fingers and disappear. The patient noticed that when the nodule is absent the swelling is larger.

Examination revealed in the second right intercostal space two fingers' breadth from the edge of the sternum a soft lense-shaped swelling three by two and one-half inches with indefinitely defined borders. It is attached to the deeper parts, the skin is freely movable over it and there are no signs of discoloration or surface heat. Pressure does not influence the size of the mass but posture does. In the erect or prone position the swelling is diminished, on bending forward the tumor quickly resumes its usual size. When the mass is shrunken a hard round body about one-eighth inch in diameter can be felt; the nodule is suggestive of a bony or cartilaginous fragment. At times it slips from the fingers and seems to disappear between the ribs. Coughing slightly enlarges the swelling, it does not pulsate, no thrill is felt and no murmurs are heard. The mass gives the impression of being a cold, or a latent bone abscess which connects with the thoracic cavity. The X-ray studies show an indefinite shadow three by four inches apparently in or just behind the anterior thoracic wall. The nature of the shadow was cleared up at operation. Plates taken after the operation failed to reveal it.

The patient was shown before the Surgical Conference and various diagnoses were made. The majority favored a cold abscess communicating with the thoracic cavity, a lipoma or a dermoid cyst communicating with the chest cavity.

At the operation a shirt-stud hæmangioma was found, the superficial lense-shaped expansion lay just beneath the pectoral fascia, the deep expansion lay between the pec-

* Read before the American Surgical Association, May 2, 1928.

toralis major and the intercostal muscles. The stem of the collar-button connecting the deep and superficial portions was composed of a dilated varix which contained a hard ivory-like phlebolith. The deep portion of the hæmangioma communicated by several branches with the vessels of the thoracic cavity. The phlebolith which was situated in the stem of the collar-button moved forward and backward like a ball-valve. When the patient sat erect it slipped backward and blocked the main communicating vessels; the same check-valve effect was produced when the patient lay on her back. On bending forward the phlebolith slipped into a side expansion and allowed a reflux of blood from the posterior to the anterior expansion. The operation was performed under local anæsthesia and the mechanism of the filling and the action of the phlebolith could be readily observed. The hæmangioma was excised "en mass" and its communicating branches to the thoracic cavity ligated. There have been no signs of recurrence and the patient has worked steadily since the operation.

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MULTIPLE PERFORATED GASTRIC ULCERS

MASSON and Simon have recently reviewed the literature of multiple perforated gastric ulcers. They were able to find records of only thirty-two authentic cases, covering a period from 1876 to the present date. In view of the rarity of multiple perforated gastric ulcers, we wish to contribute the following case:

CASE REPORT

Male, aged fifty-nine years, first seen in 1922 with symptoms indicating presence of an ulcer of the stomach; relieved by medical treatment. Five years later, October, 1927, again seen suffering from recurrence of stomach symptoms; he was still a fairly well-nourished man; increased acidity of gastric contents; occult blood in fæces; fluoroscopic examination of stomach revealed usual findings of an outlet ulcer with marked retention of barium at end of eight hours. At operation, October 29, 1927, there was revealed an ulcer in the first portion of the duodenum surrounded by large amount of scar tissue. Multiple adhesions between the pylorus, liver, and gall-bladder. The pylorus is almost occluded. Careful examination of the entire stomach did not reveal any other pathology. Convalescence normal until twenty-one days after operation when his abdomen became moderately distended. The following morning there was marked abdominal distention, and repeated enemas were not effective. The patient did not vomit. On examination, there was no board-like rigidity of the abdomen; no dulness in the flanks; no shifting dulness; slight tenderness present in the lower left quadrant of the abdomen. Patient was very weak. At this time the possibility of an intestinal obstruction was considered, and after consultation the following day, an enterostomy was made.

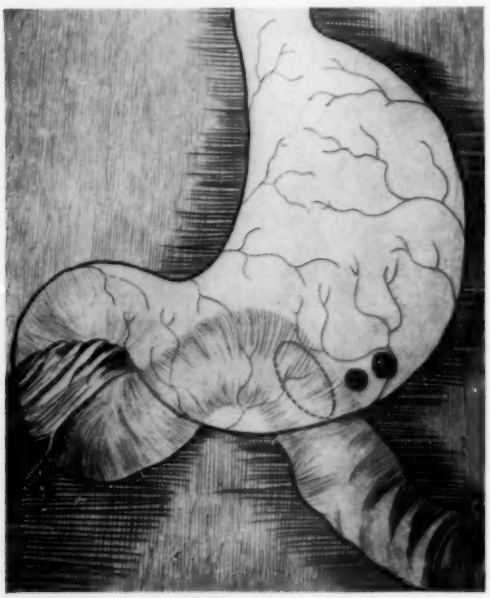

FIG. 1.—Multiple Perforated Gastric Ulcers.

A large amount of gas was obtained through the tube. There was no evidence of blood or free fluid in the abdomen. Patient returned to his room in good condition.

The following day a blood count showed a decrease in hæmoglobin to 50 per cent., with a corresponding fall in the red blood cells, and the patient was given a blood transfusion of 400 cubic centimetres of whole blood. He reacted slightly, but the next day was very weak and at times irrational. His condition became progressively worse, and he expired on November 22, twenty-four days after operation.

At autopsy there was no evidence of malignancy or obstruction in the intestinal tract. Inspection of the upper abdomen revealed the site of the gastrojejunostomy to be in excellent condition, with no evidence of leaks or adhesions. The stomach, which was three times the normal size, was found adherent to the parietal peritoneum by a large plastic exudate. Inspection of the gastrojejunostomy from the inside revealed that its condition was satisfactory. About two inches to the left of the stoma two clean-cut perforated ulcers, about one centimetre in diameter, were found situated side by side on the greater curvature posteriorly. (Fig. 1.) The stomach contents had burrowed downward and backward in the lesser peritoneal cavity, surrounding the posterior surface of the stomach and giving rise to a low-grade peritonitis found in that area. Other inspections were negative.

This case is of special interest because: (1) The stomach was carefully examined at the time of operation and the only ulcer present was located in the duodenum; (2) the patient did not have the typical symptoms of a perforated viscus; (3) the location of the ulcers, which was different from the thirty-three cases found in the literature; (4) in the large majority of the reported cases, the ulcers have been of the "kissing type," that is, one situated on the anterior and one on the posterior wall opposite each other, whereas in this case, the twin ulcers were situated side by side; (5) only thirty-three authentic cases of multiple perforations of gastric ulcers have been reported in the literature.

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THE INTRASPINAL USE OF LIPIODOL

THE use of lipiodol intraspinally is still sufficiently new to occupy an unsettled position in the group of standardized methods of spinal investigation. Opinions as to its safety, reliability and convenience are quite divergent, varying from the assurances of Sicard and his co-workers of its entire innocuousness to the complete denial of all claims made for it as a non-irritating substance and the prohibition of its use by at least one clinic in this country. Because of these facts it is obvious that the reporting of cases in which it has been used is still highly desirable for it is only by this means that the true value of the method can be determined.

FIG. 1.—Complete "block" with outline of tumor shown by margin of the oil.

In the first of the three cases reported herein two centimetres of the French preparation were injected through the lumbar route. In the second the technic was the same while in the third the only change made was to increase the amount to four cubic centimetres. A previous examination of the spinal fluid had been made in each case and had failed to reveal any evidence of meningeal irritation or infection.

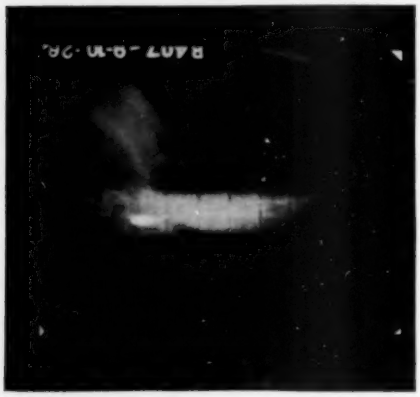

FIG. 2.—Complete "block."

Following the injection the patients were placed in the inverted or Trendelenburg position for about five minutes before the X-ray plates were made. Röntgenograms were again made at the expiration of about half an hour. It is noteworthy that in none of these secondary plates had the position of the opaque material changed. In two of the cases the X-ray examination was repeated the day after the injection, the material failing to move from its caudal position in one case, while in the other practically exact duplicates of the previous plates were obtained. In all three cases there was a slight rise in the temperature the day after the introduction of the lipiodol but on no occasion did it reach 100° F.

Operation and removal of a tumor followed in two of the cases, one within a few days and the other fourteen months after the injection of the oil.

In two of the cases lumbar puncture prior to the use of the lipiodol had resulted in a definite increase in the amount of pain the patients were complaining of. In both of these the introduction of the oil produced no greater increase than had the simple withdrawal of spinal fluid. In the third case neither the simple puncture nor the injection caused any increase in the severity of the symptoms.

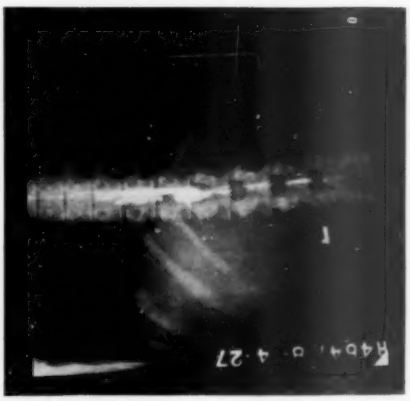

FIG. 3.—Patient in prone position to show "stringing out" of lipiodol in the absence of a "block" of the canal.

At operation no adhesions were found in either case and these three cases certainly showed no ill effects from the use of lipiodol. It served to real purpose in the third case by definitely removing the patient from the danger of having her cord unnecessarily explored. That iodized oil is not necessary for the establishment of the diagnosis in the large majority of cases is obvious, nor is it maintained that it was essential in these cases, particularly the

first two, but it should be equally clear that it is a method that has distinct possibilities in doubtful cases and is not entirely to be dismissed as too dangerous for use. After all it is certainly better to have a small amount of oil at the bottom of one's caudal sac than to have an unnecessary exploration performed or to carry a tumor that has been overlooked.

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BOOK REVIEWS

A Text-book of Fractures and Dislocations. By Kellogg Speed, M.D., Second Edition. Octavo, cloth, pp. 952, 987 illustrations. Philadelphia, Lea and Febiger, 1928.

The second edition of "A Text-Book of Fractures and Dislocations" has grown to be a volume of 952 pages with 987 illustrations. It has been thoroughly revised and much new material added.

The edition has been enriched by the lessons learned in the "Great War". The author has digested these, chosen the most practicable ones and has made them available to civil practice.

By improving the topographical arrangement the material has been made more readily available for the student. With few exceptions the illustrations illustrate. Simple-line cuts showing the basic mechanical principles underlying the treatment of fractures by suspension, and traction would have been preferable to the photographs crowded with forests of ropes, pulleys and weights. The graphic representations of the demonstration booths of the Fracture Committee of the American Medical Association are novel and serve a purpose. The movie-like catch phrases coupled with the illustrations appeal to the visual sense, fixing the ideas better than the written or spoken word. Fig. 232, page 307, and Fig. 677, page 665, of these demonstrations are misleading. The resulting forces shown are not such as conduce to mechanical efficiency. The reviewer understands that the same faults were evident in the actual demonstrations.

The chapter on the general treatment of fractures is sound and full of invaluable advice on what to do and what not to do. The chapter devoted to "The Operative Treatment of Fractures in General", is an able, practical, and liberal exposition of the underlying principles. The chapters on the various fractures and dislocations of individual bones and joints are remarkably free from fads and fancies. The treatment and after-treatment of the various lesions are presented with a fulness balanced by a proper regard for detail. The advice offered is sound and full of common sense.

It is impossible to please everyone and keep a text-book within practical limits. Nevertheless, we wish that Doctor Speed had allowed us to share with him his rich pathological experience. We also feel that the work would have been enhanced by a suitable historical background. This feature is one of the charms of Stimson's classical work. A knowledge of what the pioneers in our craft have done and what we owe to them should be readily accessible to all students. The necessary information should come to them in connection with the subject in which they are working. It should not be hidden away in a disconnected history. Such knowledge enhances

the morale of the profession and stimulates the individual to be something more than a mere automaton.

Throughout this monograph a liberal point of view is taken and there is no attempt to throttle ideas by the popular canker of dogmatic standardization. It mirrors the modern American practice of the treatment of fractures and reflects the ripe experience of a surgeon well versed in traumatic surgery.

H. H. M. Lyle.

Sinus Thrombophlebitis: Inflammatory Diseases of the Venous Sinuses of the Dura Mater. By Alfred Braun, M.D. 4to. Cloth, pp. 269. New York, Paul B. Hoeber, 1928.

This work is a fairly complete summary of the subject of sinus thrombophlebitis, but naturally, except for the anatomy, deals chiefly with the sigmoid sinus. The first third of the volume is devoted to anatomy, with a small section on embryology and development. Detailed descriptions of all the venous sinuses are given and the numerous variations described. The following third is devoted to pathology and symptomatology. A detailed review, in order, of all the various symptoms is given, and in many instances statistics of various authorities are quoted. However, some are rather lightly passed over, as where the author states "that there is an increase in the total number of leucocytes" notwithstanding that this is not always met with and that there is frequently a leucopenia in children.

The last third is chiefly devoted to the treatment of sigmoid thrombosis. The author describes the standard operation with complete removal of the sinus wall and thrombus, but makes little mention of the variations from this which are now advocated by many otologists. The subject of cavernous sinus thrombosis, both as to symptoms and treatment, is rather briefly reviewed, with frequent reference to Eagleton's work.

Throughout the volume the author rarely mentions his own experiences, and for some reason quotes none of his own statistics, thus giving the volume the aspect of a general and fairly complete review of the subject, rather than a personal authoritative work. Thus the Tobey test, with its modifications, is described, but it is left entirely to the reader to judge if the procedure may be of value.

The volume is profusely supplied with original and well-selected illustrations, which add greatly to the text. As a general survey, the work should prove of value to any otologist both for study and for reference.

Robert L. Moorhead.

Proctology. A Treatise on the Malformation, Injuries and Diseases of the Rectum, Anus and Pelvic Colon. By Frank C. Yeomans, M.D. D. Appleton & Co., New York, 1929. Large 8vo; cloth; pp. 661.

In this book, which bears the date of 1929, the effort of the author has been to present a practical view of the knowledge of to-day in the affections

of the particular section of the body to which it is devoted, namely, affections of the lower colon, of the rectum and of the anus. The result is worthy of commendation for the book as a whole covers well the field of surgical effort which the progress of later years has been made to become very large and important.

The systematic treatment of the various subjects involved has been sufficiently detailed to swell the size of the book to a very marked degree and yet examination of its more than six hundred large octavo pages does not show anything which might have been omitted without a distinct loss. The chapters on anatomy, physiology, embryology and developmental defects are excellent and are fundamental. The chapters upon the examination of the patient are thorough and instructive. One cannot help a smile of amusement, however, in noting that in the illustrations devoted to sigmoidoscopy, the examiner is depicted with his face thoroughly protected by an aseptic mask and one wonders whether the use of such a mask at such a time was intended to prevent possible infection of the rectum by nasal secretions or to protect the face from the possible results of too forcible rectal explosions. To say the best, the mask does no harm, anyway.

The great field of the proctologist is undoubtedly that pertaining to the relief of hemorrhoidal conditions. It is to be wished that the anatomy and etiology of these conditions had been more fully elaborated. When it comes to the treatment of hemorrhoids, the author declares himself as most favorable to the clamp and cautery method, saying that he "employs it more than any other operative method and with most satisfactory results". We must confess some surprise at this for we had thought that the trend of more recent surgery had been to send even the hemorrhoidal clamp and cautery back to association with the cauterizing irons of the general surgeon of the days of Paré and to substitute therefor the more ideal methods of direct excision and immediate suture. What little suggestion the author makes toward the latter methods are imperfect and unsatisfactory. This criticism, however, should not prevent one from an appreciation of the value of the many other chapters of the work.

LEWIS S. PILCHER.

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